

THE IMPACT OF INTRODUCED DISEASES IN
THE PRE-TREATY PERIOD 1790 – 1840

A Thesis
submitted in partial fulfilment
of the requirements for the Degree
of
Masters of Arts in Maori
in the
University of Canterbury
by
D E Hanham

University of Canterbury

2003

Table of Contents

	Page
Abstract	1
Introduction and Literature Review	2
Chapter One - Disease and Endemicity in the Pacific	14
Chapter Two - Diseases Genetic or Endemic to New Zealand	32
Chapter Three - The Impact of Acute, Infectious Introduced Diseases	45
Chapter Four - Venereal Diseases and Tuberculosis	61
Chapter Five - Maori Values and Beliefs Regarding Introduced Disease	78
Conclusions	92
Acknowledgements	98
Bibliography	99

ABSTRACT

This thesis explores the impact of infectious introduced diseases on pre-Treaty Maori society. It addresses significant gaps in the current literature including consideration of the Pacific context from a microbiological perspective and modern analysis of an inadequate primary New Zealand literature on which present views of Maori health rely. The premise of the thesis is that few diseases could have been imported into New Zealand with the initial Polynesian immigrants, and that the impact of European-introduced infectious diseases would have been greater than has been previously realised. The thesis evaluates the incidence of introduced infectious diseases, including the development of immunity, and the impact on Maori beliefs about causality and transmission, and in particular the question of whether the attribution of lung disease to an entrail-eating atua was a post-contact development. The thesis argues that rather than, as previously thought, a wholly pre-contact concept, the idea of the flesh eating lizard god was an attempt to comprehend tuberculosis, and was specific to tuberculosis. The thesis further argues while Maori initially blamed many of the diseases on 'European atua', reflecting the multiplicity of gods in their own culture, as interaction with missionaries increased the blame began to swing to the European God, and his priests, the missionaries. In response to the perception of the power of this god Papahurihia of Northland developed an anti-missionary doctrine based on biblical ideas in which the serpent of Genesis is revealed as the most powerful god. This, it is argued, is a development of the association of lizard gods with introduced tubercular disease.

INTRODUCTION AND LITERATURE REVIEW

The indigenous social history of medicine in New Zealand's pre-treaty period is a topic that has received very little attention from scholars. The literature leaves a number of significant issues to be addressed. The topic is sparsely recorded in the primary European sources, and at that by people who had little knowledge of Maori culture. The purpose of this thesis is to take a fresh look at the earliest available European observations of Maori health in the 1790 - 1840 period. The contemporary literature will be examined in this thesis in the light of scientific, microbiological and historical resources to determine the impact of introduced diseases on Maori in terms of morbidity and mortality in the early contact period. The thesis sets out to explore the possibility that this impact would have been greater than has been previously realised. While the impact of introduced diseases on established beliefs and practices would require a full-scale examination of traditional society that is outside the scope of the present study, the question of whether Maori reinterpreted traditional concepts in order to enculturate introduced disease, in particular, tuberculosis, will also be examined.

While there are brief descriptions of health and disease throughout both the primary and secondary literature on New Zealand, there have been limited attempts to draw this evidence together into a comprehensive body of work. Consequently, the available literature is characterised by four significant limitations. First, there is a lack of scientific analysis that determines whether diseases were endemic, introduced or hereditary. Second, there is no contextual Pacific background to determine whether diseases were endemic or introduced, and if introduced, from where. Third, there is no analysis that traces the introduction of infectious diseases to determine their impact on morbidity and mortality. Thus there is a need to determine which diseases were endemic to New Zealand before the arrival of Europeans. There is also a need to determine the impact introduced diseases would have had on morbidity and mortality. Fourth, most secondary authors have tended to take the primary literature at face value as a static and generic picture although it deals with an approximate 40 year period of which rapid change is characterised.

The primary data is widely disseminated across a variety of journals, diaries, ships' logs and letters written by visitors to New Zealand such as Dr Savage (1805), R A Cruise and Dr Fairfowl (1820), E Markham (1833), Dr Marshall (1834) and long-term residents such as Joel Polack, a trader in the Bay of Islands for several years. This creates a piecemeal body of information that needs to be assembled like a jigsaw puzzle, comparing each piece of evidence against other pieces to find out where exactly it should fit.

Most of the Europeans who visited New Zealand did not come with a benevolent anthropological desire to study Maori society and therefore the literature does not comprise a detailed observational study. The primary literature is written mostly by casual observers who came to New Zealand with a specific purpose such as commerce, colonisation, or to pursue an interest in science, or by long term residents such as the missionaries who came with the intention of converting Maori to Christianity. All of these reasons have an impact on what they would have recorded. For instance, some evidence was recorded by men who were in New Zealand for commercial pursuits such as whaling, sealing and trading. Therefore many of the journals focus on day-to-day activities and only passing mention is made of ill health with regard to the loss of industry. The most that can be gained from these journal entries is an indication of dates, times, places where illnesses were occurring. Many eyewitnesses were in New Zealand for only a very short period of time and offer only passing mention of diseases or epidemics at a given time or location. Some of the more comprehensive literature was also recorded with a specific purpose in mind. For instance, men like Dr Ernest Dieffenbach and Dr Edward Shortland did survey Maori health more specifically, but their purpose was to ascertain the health risks for colonising Europeans. Dieffenbach recorded a significant amount of data in the late 1830s, however, he stated from the outset that he was writing as a naturalist for the New Zealand Company. His purpose was to inform his readers of the possibility of colonising New Zealand by giving his observations of the flora, fauna, climate and health of New Zealand. (Dieffenbach 1843, p iii - IV) Similarly, the Colonial Government of New Zealand employed Edward Shortland as Protector of Aborigines and an interpreter. Shortland was the only medical doctor up to 1840 who had observed Maori health in the South Island. However, he was also assessing Maori health for colonial purposes. As many eyewitness accounts are influenced by the

eyewitnesses' purpose for recording the evidence or giving of evidence, caution is needed in assessing the data. All of the primary literature, regardless of whether it was written by medical professionals or not, was written before there were any major advances in medical science, such as the discovery of micro-organisms. These limitations have resulted in a number of contradictions, conflicts and inaccuracies in the primary data that have not been widely addressed by the secondary literature, such as claims epidemics had not occurred when the evidence showed they had; or eyewitnesses who made observations but contradicted themselves in other statements. These issues will be discussed in more detail below, as they underpinned the approach taken in managing the evidence.

In addition, the secondary authors, such as H. Wright (1959), L. K. Gluckman (1976) and R. Lange (1999) all acknowledge that this field of research still requires a comprehensive study. For instance, Gluckman comments:

The medical history of early New Zealand is a history of some complexity. Much of the literature has yet to be systematically studied or indexed. Terminologies and theories of disease change and what is written at a given date can only be evaluated in the light of the theories of disease at that date. The only approach available for the medical historian is the approach of the widespread reading of original documents, journals and other papers. (Gluckman 1976, p 15)

Gluckman also commented that a thorough review of ships' logs and journals of ships' doctors would be a beneficial contribution to New Zealand's medical history, (Gluckman 1976, p 100) as New Zealand was visited by a number of naval vessels from 1830 – 1846. In addition, journals, eyewitness accounts, letters and diaries all require reviewing. There is therefore a need to collect, collate and analyse as much of the literature as possible.

This study necessarily relies on European observations of Maori rather than explanations of Maori by Maori, as there is a shortage of Maori literature for the period. The European accounts yielded a significant body of material regarding some Maori beliefs and practices, which have not been thoroughly addressed by the

secondary authors. Some of the missionary descriptions of Maori inadvertently give valuable insight into Maori beliefs and values; however, the evidence was not always recorded for that purpose. As an example, missionaries such as Henry Williams recorded that Maori accused the missionaries of bringing diseases amongst them, although the missionaries scoffed at such an idea. This missionary evidence suggests that Maori were developing a theory of disease transmission and is therefore a valuable insight into how Maori were thinking. Nonetheless, a significant difficulty in much of the secondary literature is the lack of analysis of Maori perspectives. As the research progressed it became more and more apparent that Maori perspectives with regard to health needed to be thoroughly reviewed. Although a linguistic study would provide some insight into practices and beliefs, and perhaps morbidity and mortality, it is sufficiently complex as to warrant a study of its own. While Maori were beginning to read and write from the late 1820s, there is no substantial pre-Treaty body of Maori writing. Reverend Yate provided translations of a number of letters written to him by Maori in 1835, however, the Maori text is not included and what words were used by Maori to describe symptoms and diseases cannot be identified. Joel Polack, a trader who lived at the Bay of Islands for several years and recorded his observations of Maori, also commented that he had a number of letters in his possession written by Maori, however, the whereabouts of these letters is unknown. Other material, such as waiata, myths and legends, that were collected by George Grey after 1840 have yielded no information on diseases. The conclusions that can be drawn from this is that Maori either did not have these diseases prior to European contact or that Maori did not focus their myths and legends around diseases.

The medical and scientific data in the literature reviewed requires a deeper and more contemporary analysis, than the contemporary accounts can provide. When observations of Maori health were written between 1790 – 1840 medical science was in its infancy. The miasmatic theory, which views disease causation as environmental, was the dominant theory of the period. The germ theory, which is still popularly held today, did not begin to have any serious credence until after 1840. The paucity of medical and scientific analysis continues into the secondary literature and limited attempt has been made to address the origins of disease, the spread of diseases into New Zealand or to ascertain which diseases were introduced and which diseases

were endemic.¹ Consequently there are large gaps in the literature. In the last fifty years scientific and medical knowledge has increased significantly. Clinical diagnosis of disease is more accurate and comprehensive, the classification of diseases is more specific and more recent scientific methods allow us to access information that was unknown, inaccessible or under utilised before. This knowledge and technology provides tools to help analyse the diseases that occurred in the pre-Treaty period.

There is a need for a more specific, forensically researched and analysed body of literature, in order to reduce or eliminate some of the discrepancies in the primary literature. An analysis of which diseases were endemic or introduced in the Pacific region and therefore carried into New Zealand by the initial immigrants would be beneficial. From that point, an analysis of what diseases may have been genetic and what the state of Maori health was prior to the impact of introduced diseases would help to assess what impact the epidemics and diseases would have had on morbidity and mortality and on beliefs and practices. Many of the smaller gaps in the literature have arisen as a result of the primary literature not being collected, collated and analysed as thoroughly as it could be. The problems described above were therefore exacerbated and our current understanding of the medical history of New Zealand prior to 1840 requires a thorough review to address a number of misconceptions that may have arisen from the information currently available. This thesis attempts to begin that process.

The introduction of acute epidemic or other contagious diseases into New Zealand was facilitated by the interaction between an immunologically naïve indigenous population and visitors from around the globe. It was therefore necessary to look at first contact to establish, as far as possible from the observational data, what the state of health was for the indigenous population, what diseases were introduced, and where possible from where they were introduced. As the first eyewitness accounts from early explorers such as Captain Cook, Jean de Surville and their supercargo and crew did not offer a complete picture of Maori health at first contact a brief review of the history of Pacific Island health was embarked upon. The analysis of the history of Pacific Island health also provided a microbiological and scientific analysis of both

¹ Diseases do not simply arrive or manifest by themselves they must have a cause whether it is genetic or

endemic and introduced diseases in the Pacific. Microbiological works, such as Dr Miles' *Infectious Diseases: Colonising the Pacific*, were used as evidential literature on how micro-organisms affected human health in the Pacific and to assist the understanding of the epidemiology of introduced diseases. A microbiological analysis of the diseases can be useful in identifying whether the diseases were introduced or not and what impact the diseases would have had.

The issue of whether the diseases in question were zoonoses, diseases of animal origin, has not been considered in any depth. As Maori did not have any domesticated animals, aside from the dog, many of the infectious diseases, which were mostly zoonoses, might not have existed prior to contact. A microbiological analysis may also help to identify diseases specific to Maori that affected Europeans on contact. This is research that has not been undertaken at all in New Zealand. By tracing endemic and introduced diseases from Melanesia through West Polynesia and into East Polynesia it is possible to establish that a number of diseases may possibly have migrated to New Zealand with the initial immigrants. Having established what infectious diseases may possibly have migrated to New Zealand an analysis of Maori health from archaeological records and eyewitness accounts was undertaken. Phillip Houghton's *The First New Zealanders* provided the bulk of the archaeological evidence. The eyewitness accounts of Maori health include background evidence from 1769 - 1790 to help establish the state of Maori health in the absence of infectious diseases introduced by Europeans. The majority of the literature in the three final chapters on introduced disease and cultural influences is taken from ships' logs and journals and the records of travellers, traders and missionaries. An analysis of these sources has been used to create a New Zealand literature on the introduction of acute and chronic diseases. It had been hoped that the focus of the thesis would concentrate on the South Island and lower North Island with reference to the sites of early contact in the upper North Island. These regions had not been studied with regard to the introduction of diseases and it was hoped a focus on these regions would widen the field of knowledge, as the available secondary literature is strongly focussed on the upper North Island. Unfortunately, there is little evidence for these regions recorded prior to 1840.

The collection, collation and analysis of the primary literature highlighted a number of difficulties, such as accessibility, conflicting or contradictory evidence and misdiagnosis. A thorough review of the primary literature was complicated by accessibility. While there is a large body of published material, it sometimes yielded very little information, however, there is also a significant body of material that is in manuscript form and inaccessible. Accessibility was also difficult because of the age of the materials. While the item itself might be available the evidence within it was often difficult to access. The older-style books lacked indexes or were poorly indexed and each item had to be read throughout to ensure that evidence was not overlooked. While this was a tedious process it often revealed small pieces of information that were nevertheless of considerable significance. Although a large body of evidence has been accessed in this manner, there are still a number of areas where the evidence is inevitably thin.

Conflicting evidence required careful analysis. There were often a number of reports of an incident, epidemic or people who were unwell, however, each eyewitness might have a different point of view or provide conflicting evidence. It then became necessary to decide who was most likely to be speaking with the most authority, if any, to get as close to the truth as possible. This method could only be applied to each issue on a case-by-case basis, as each individual might be an authority unto themselves in a variety of ways. Therefore, while a medical doctor might have greater authority in identifying diseases, within the restrictions of the era, someone who had lived a number of years amongst Maori might have greater authority in other matters. Who was most authoritative in any given situation was dependant on the issue being discussed. As an example three people off the *Dromedary* all reported different information on the incidence of venereal disease. One said there were some cases, one said 14 – 15 cases, and another said it was prevalent. One of these people was Dr Fairfowl. Presumably, as a medical doctor, his statement of 14 – 15 cases was the most accurate. Therefore, it became clear that conflicts in the evidence need to be addressed more thoroughly than has been done to date. In addition, conflicts with the evidence often arose because the evidence was restricted solely to the time period, or location where the eyewitness was in New Zealand. For example, in 1834 Dr Watkins noted that there were no smallpox or measles. (Watkins 1908, p 20)

However, an epidemic of measles did occur in the South Island and an epidemic of smallpox may also have occurred very early in the contact period. As Watkins was in the North Island only briefly he would not have been aware of any epidemics before or after his arrival or in other locations in New Zealand. Therefore, statements such as these cannot be taken at face value. Contradictions also arose when eyewitnesses did not clarify their comments. For instance, Polack claimed that epidemic diseases did not have a significant impact on Maori. (Polack 1976, p 97) Polack also commented that New Zealand was free of the epidemics of dysentery, measles, consumption and diarrhoea that afflicted New Holland, (Polack 1838, p 360) although Polack himself had earlier mentioned an epidemic of measles and many deaths from consumption. If, as Polack claims, New Zealand was free of epidemic disease, logically New Zealand would be free of the impact. However, during the period that Polack was in New Zealand a number of epidemics of respiratory diseases occurred. Presumably, Polack meant the epidemics he observed did not have an impact, not epidemics in general.

Not all locations suffered simultaneous epidemics. Nonetheless, evidence from specific localities has previously been applied or implied generally across the country. This creates the impression that all Maori were affected at the same time with all the same problems, but this is incorrect. Not all areas of New Zealand were frequented by Europeans, and some areas were frequented more often than others. Therefore the impact of diseases would differ across the country. The literature tends to focus on Maori health in the North Island, especially the Bay of Islands; however, from the early 1790s there were a number of ships at Dusky Bay and around the southern South Island where sealing was based. Evidence in Robert McNab's *Murihiku* records that diseases may have been well established by the early 1800s. This would suggest that the diseases were introduced at an early stage. It is therefore important to be specific, where possible, about the location the evidence pertains to. Similar misconceptions have arisen regarding the time period in which epidemics occurred. By 1840 Maori society had undergone a number of significant changes. However, the degree of change varied across the country as a result of the different degrees of contact. To understand what impact introduced diseases would have had we need to understand the pattern of epidemics, and this needs to be done decade by decade. It is therefore necessary to find records of the earliest possible contacts. Some of the

current literature is heavily reliant on evidence from the post 1820 period, especially evidence recorded by missionaries, leaving gaps in the literature from 1790 to 1820. By the 1820s, especially in the Bay of Islands, missionaries and other Europeans were well established and so were a number of diseases. Missionaries were not present in the lower South Island until the 1840s although whalers, sealers and traders were. Where missions were established there was a direct influence on beliefs and practices, which had become increasingly influential by the mid to late 1830s. Therefore, while it is possible that diseases were introduced at similar times the impact on beliefs and practices in the two locations might be very different across different time periods. Using evidence from a later period may give a false impression of the situation at the time. It is therefore important to review the literature from the stated period and location.

The issues discussed above have led to a number of generalisations in the literature that require clarification. For instance, there are numerous comments that skin diseases were common amongst Maori. However, skin diseases appear to be introduced or symptomatic of other introduced diseases. Skin diseases may have become common by the 1830s, most especially in the Bay of Islands, but a sweeping generalisation that Maori were affected by them fails to address the fact that the diseases were probably not endemic. When the first Europeans arrived in the late 18th century there were a number of reports of Maori free from skin diseases. One observer, L'Horne, who was at the Bay of Islands on the first French vessel, made comment that a few Maori had sores and ulcers, but these people had already been in contact with Europeans and the diseases were possibly introduced at that point. There is a lack of evidence to suggest skin diseases were pre-European. Therefore, it seems clear that generalisations can distort accuracy and need to be minimised.

Diagnoses were sometimes inaccurate and described a symptom rather than a specific illness, as eyewitnesses' often tended to describe illnesses from their own perspectives. This could be problematic as it would not be acceptable to re-diagnose the cases without reasonable evidence. However, as an example, Kendall showed a tendency to describe tuberculosis as 'a violent cold'. This is rather an understatement. Given that other missionary evidence showed that the people Kendall described as having 'a violent cold', such as the famed case of Ruatara, later died of tuberculosis,

it is reasonable to redefine Kendall's diagnosis. This is also true for tumours, abscesses, pustules or boils these seem to be interchangeable terms in the observational literature and need to be addressed in the context of other symptoms, or diseases that may be prevailing at the time the descriptions are given.

The issues raised above have resulted in the adoption of a forensic method of analysis, which has resulted in a number of instances, in new evidence coming to light regarding the medical history of New Zealand. This combined with an historical and scientific theoretical approach has resulted in an overview of the period from a number of new perspectives, thus justifying the need to readdress the currently available literature. However, the evidence of all these sources can only provide an overview of Maori health and the impact of introduced disease. As the thesis has progressed more and more sources have come to light and this thesis therefore is a platform for further research.

AN OVERVIEW OF THE CHAPTERS

Chapter 1: Infectious Disease and Endemicity in the Pacific

Large gaps in the literature meant it was necessary to trace Maori origins through the Pacific and into New Zealand to assess what diseases were either endemic or introduced by Europeans. By identifying the diseases that may have been endemic to the Pacific it was possible to identify what diseases might have been brought into New Zealand by the initial immigrants and were therefore endemic to New Zealand. This would also help to identify what diseases were introduced into New Zealand by Europeans. Genetic evidence of Pacific migration was helpful in establishing the movement of people through the Pacific and assisted in identifying what diseases may have been genetic to New Zealand Maori. However, the range of diseases that could be assessed was restricted to the range of diseases that have been researched by experts such as microbiologists and archaeologists, although that field is relatively wide.

Chapter 2: Diseases Genetic or Endemic to New Zealand

It appears from the evidence that the range of infectious diseases or disease causing micro-organisms that were endemic to New Zealand was very small, although a range of genetic diseases, such as cancers, kidney and heart disease, did exist. The range of

micro-organisms capable of causing disease that were possibly endemic were restricted to strains of streptococci and staphylococci, although the evidence for their existence in New Zealand is slight, and the viruses responsible for hepatitis B and herpes, although the evidence for their existence is also slight. With a low incidence of infectious diseases Maori would have been reasonably healthy and their reasonable good health was reflected in their good physique, excellent healing ability, good teeth in most regions, despite later wear and tear, and longevity. All of this changed with the introduction of acute infectious epidemic diseases.

Chapter 3: The Impact of Acute, Infectious, Introduced Diseases

It would appear that some diseases had a greater impact on Maori health than others. Diseases such as dysentery, mumps, whooping cough and polio could potentially have had a devastating impact on an immunologically naïve population. However, although a number of deaths occurred the overall morbidity and mortality rates were not incredibly high, and more importantly appear to have been largely restricted to the Bay of Islands and upper North Island. However, an epidemic of measles in the South Island had a devastating impact that affected the whaling industry, which was reliant on Maori labour, but later led to whaling stations being established on land once occupied by Maori. This effectively altered the balance of power. Catarrh and influenza epidemics up and down the country in 1820 and 1827 caused a number of deaths in some regions. Maori in Taranaki claimed that thousands died in the 1820 epidemic; however, in 1827 the death rate does not appear to be that high. The epidemics in 1836 - 1838 appear to have had a great impact with significant mortality, especially amongst the old people at the Bay of Islands.

Chapter 4: Venereal Disease and Tuberculosis

The diseases that affected Maori most of all were tuberculosis and venereal diseases, especially syphilis. These diseases impacted not just on morbidity and mortality but also on some beliefs and practices regarding disease. The wars of the early 19th century saw a large slave population established in the Bay of Islands. The girls and women were put into sexual slavery. According to the evidence they were used for the shipping industry and put under tapu as a result of venereal disease, probably syphilis. A number of measures were used by the women to reduce the impact of syphilis, such as a vapour bath using specially selected herbs. The practice of

infanticide or abortion was common amongst these women and would have resulted in a reduced incidence of congenital syphilis, although this does not appear to have been the goal of the practice. Tuberculosis appears in the literature from around 1808 and by the 1830s was having a hugely significant impact on morbidity and mortality and also on beliefs and practices. Tuberculosis manifested in many forms, however, scrofula and pulmonary tuberculosis were the most common.

Chapter 5: Maori Values and Beliefs Regarding Introduced Diseases

Atua, which had been familial were now believed to be European atua and debate arose amongst Maori as to the exact cause of the disease. A number of theories arose, such as a theory of disease transmission either from ships or directly from people, but the theory that gained the most attention was the belief that missionaries were he iwi makutu, sorcerers. This theory gained a following and appears to be one of the major underlying reasons for the development of the cult of Papahurihia. This cult was the first cult of its kind to appear in New Zealand and it would seem it arose as a reaction to a combination of Christian teaching and infectious diseases, that were linked in Maori minds by the teachings of the missionaries. The introduction of infectious diseases therefore brought about fundamental changes in Maori beliefs regarding disease.

CHAPTER 1: INFECTIOUS DISEASE AND ENDEMICITY IN THE PACIFIC

Any disease endemic to New Zealand Maori in the pre Treaty period would also have been endemic in the Pacific and most especially in Eastern Polynesia. Therefore, a study of infectious diseases in the Pacific and especially eastern Polynesia will provide a contextual background. A number of methods can be used to assess this. One is to look at the Pacific region to trace the migration of Polynesian people and diseases from their origins to their eventual arrival in New Zealand. There are two factors to be considered with this method. First, the diseases themselves - where certain infectious diseases may have originated from and how they affect human health - and second, the migration of people and carriage of disease. By establishing migration patterns it is possible to gain a clearer insight into the possible carriage, spread and endemicity of diseases. However, many diseases would not have established themselves widely across the Pacific, as the likelihood of a disease establishing itself is dependent upon the disease. Diseases relevant to this study can be split into two main groups: genetic diseases and infectious diseases. Genetic diseases are passed on through heredity, whereas infectious diseases are transmissible via other modes such as saliva droplets, person to person contact, or contact with infected matter. Genetic diseases fall into a number of categories such as cancers and kidney disease. However, for the purposes of this research, genetic studies have more importance in tracing migration across the Pacific. Infectious diseases fall loosely into a number of categories such as arthropod, chronic or latent, and acute. The category of these diseases determines their ability to establish endemicity.

Genetic Evidence of Polynesian Origins

Genetic studies that were conducted in the 1980s have shown that genetic material and archaeological studies indicate similar migration patterns. The genetic material supports the argument that Polynesians are of South-East Asian origin. While the genetic data have not identified an exact homeland for pre-Polynesians, the genetic evidence does suggest a South-East Asian origin prior to Mongolian expansion, as Polynesians lack the alleles typical of modern Chinese. Therefore, South-East Asians and Polynesians share genetic material that acts as anthropological markers.

(Serjeantson 1989, p 286) A specific type of mitochondrial DNA is found in coastal Melanesia (8 – 42%) but not in Highland Papua New Guinea or Australian Aboriginals, although it is universal throughout Eastern Polynesia and occurs in 20% of East Asians and Japanese. (Serjeantson 1989, p 287) This would suggest a gene flow from South East Asia to Coastal Melanesia. This genetic pattern is compatible with the theory of Lapita people settling among Western Melanesians and then expanding out to Vanuatu, New Caledonia and Fiji where they were joined by new groups of Melanesian migrants. (Serjeantson 1989, p 287) The fact that pre-Polynesians lack certain genetic markers found in some coastal New Guinea areas indicates that pre-Polynesian groups did not interbreed with people in this region, but passed quickly through to other regions of Melanesia. However, some genetic markers in Polynesians show significant interbreeding in other regions of Melanesia, such as Northern Island Melanesia, (Serjeantson 1989, p 287) indicating a period of settlement and integration, which would also entail the sharing of genetic and infectious diseases.² Therefore, the settlers came from certain areas of Melanesia and spread out into Western and then Eastern Polynesia.

The number of diseases that could be transported into Polynesia was dependent on the size of the founding group. The number of genetic diseases that were carried into Eastern Polynesia would have been relatively small as the genetic and archaeological material indicates that the founding group was small. (Houghton 1980, p 73) (Serjeantson 1989, p 288) (Bellwood 1989, p 44) Serjeantson argues that Eastern Polynesians show significant homogeneity as through the process of genetic drift some gene markers have become more common. (Serjeantson 1989, p 288) This would indicate that a number of genetic diseases would be common to the Eastern Polynesian group. It may go some way to explaining the high susceptibility to diseases such as diabetes. There is some debate as to the immediate origin of the Eastern Polynesian settlers and Serjeantson argues for Samoa as the origin, (Serjeantson 1989, p 288) while Bellwood argues for the Marquesas. (Bellwood 1989, p 44) The evidence is not conclusive either way; however, the evidence for a small gene pool is supported by the development of a particular shape of skull, jaw and

² There has been some debate as to the origin of the Polynesian settlers; however, there is no indication of gene flow into Polynesia from South America and there is no evidence of a genetic link between Polynesia and Micronesia.

teeth that while not totally universal, is almost unique to Polynesians, including Maori. This Polynesian jaw is known as a 'rocker' jaw due to its rounded outer edges. The 'rocker' jaw is found in 70 – 80% of the Polynesian people, whereas in other populations it is found in only 1- 2% of people. (Houghton 1980, p 44 - 54) Therefore, there are strong indicators for a small founding group into Polynesia and consequently there would be a limited pool of diseases, both genetic and infectious.

Were Infectious Diseases Established in Pre-contact Polynesia

There appears to be a very limited range of infectious diseases that were endemic to Eastern Polynesia. The low incidence of infectious disease can be attributed to two factors: environmental - the environment in Eastern Polynesia is not conducive to a high incidence of endemic disease; and migratory - the further East the Pacific Island people travelled the lower the incidence of infectious disease. Infectious diseases affecting Polynesians would have fallen into two classes; those specific to the people themselves and those shared with their domesticated animals. Most if not all of Polynesia falls beyond the line known as the Wallace line. This line defines the boundary between lands with abundant flora and lands with abundant fauna. Most of Polynesia has abundant flora and birds, but little other fauna. Diamond comments:

Diverse epidemic diseases of humans evolved in areas with many plant and animal species suitable for domestication, partly because the resulting crops and livestock helped feed dense societies in which epidemics could maintain themselves, and partly because the diseases evolved from germs of the domestic animals themselves. (Diamond 1998, p 86)

As discussed above the founding group in Polynesia was small and this was probably also true of each wave of founders in each island group. Therefore, there were limited possibilities for endemic diseases in Polynesia due to a shortage of domesticated species and a small, transient population base. However, the founding settlers brought with them dogs, chickens and pigs, (Diamond 1998, p 60) although not all survived in all regions. While not classed as a domestic animal, the Polynesian rat also accompanied the colonisers; whether its transportation was deliberate is uncertain. In addition, the linguistic evidence suggests Polynesians had a long

association with these animals and a long association with head lice. (Diamond 1998, p 209) All of these species have been identified as capable of transmitting diseases to human beings; however, the evidence for the transmission of certain diseases to human beings has previously come from outside the Pacific. It is not therefore certain, that diseases carried by these animals in Europe were carried by the same animals in the Pacific, as it can be difficult for diseases to establish themselves.

Due to the characteristics of some infectious diseases, not all diseases would have spread into Eastern Polynesia. Infectious diseases fall into a number of different categories and the category of disease is a determinant factor in its spread and endemicity. The three categories of infectious diseases relevant to this study are: arthropod diseases, which require a vector (an intermediary agent, such as fleas or mosquitoes); chronic or latent diseases such as the herpes viruses where humans are long term carriers; and acute infectious epidemic disease. All these categories have been observed in the Pacific; however, they were not endemic to all regions.

ARTHROPOD DISEASES

Arthropod diseases, which are viral, do not appear to have spread far beyond Melanesia. These viruses are difficult to establish and would need reasonably prolonged contact as the diseases have a 'wildlife (sylvatic) cycle between arthropods (insects, ticks, crustaceans) and birds and mammals'. (Miles 1997, p 16) Miles comments that 'even if humans brought infected arthropods (vectors) with them there would not have been a sufficient susceptible population of vertebrates to establish an enzootic focus'³. (Miles 1997, p 16) Contemporary examples show that enzootic foci have failed to establish widely. (Miles 1997, p17) The range of arthropod diseases reported in the Pacific region include, arboviruses, malaria, typhus and filariasis.

Arboviruses

Arboviruses⁴ do not appear to have established in Eastern Polynesia. Most are not dependant upon humans as a host, aside from dengue fever, which has humans and primates as its host. (Miles 1997, p 94) They produce strong immunity but cannot

³ An enzootic focus is effectively a host species.

⁴ Arboviruses are a group of viruses that affect arthropods and vertebrates and have a biological cycle in both. New Guinea has a large variety of arthropods and there are a large number of vertebrates established there.

survive in a small community, as they need a steady supply of hosts. As they have a limited number of suitable hosts they have difficulty establishing themselves in a new environment. (Miles 1997, p 94) Those that are pathogenic to humans are the mosquito flavivirus, Murray Valley encephalitis, and perhaps also Japanese encephalitis virus. Both of these cause severe encephalitis. (Miles 1997, p 94 - 5) Murray Valley encephalitis and the alpha virus, Ross River virus are enzootic in New Guinea and perhaps the Solomon Islands, but not elsewhere. (Miles 1997, p 17) The Ross River virus causes poly-arthritis and is probably endemic to the Solomon Islands. (Miles 1997, p 95) There is no evidence to suggest that arboviruses are endemic in other Pacific Island groups, although epidemics of dengue and poly-arthritis do occasionally occur. (Miles 1997, p 95)

Malaria

Malaria (*Plasmodium vivax*) appears to be prevalent through most of Melanesia as far south as the New Hebrides. (Howe 1984, p 48) Arthropod diseases such as malaria, where humans are a definite vertebrate host, may have been in existence in some regions of the Pacific when Europeans first arrived during the 16th century. (Miles 1997, p 19) The vector mosquito *Anopheles farauti* had migrated from Asia into New Guinea and the Solomon Islands. (Miles 1997, p 21) Tertian and sub tertian malaria is currently present in New Guinea, the Solomon Islands and Vanuatu, with the exception of Futuna. The disease has become established where the vector, the mosquito, has become established. Although there is a possibility that malaria, the disease and the vector, did not spread into Vanuatu until after the early 1600's, (Miles 1997, p 25) the vector and the disease have not spread further into the Pacific. (Miles 1997, p 21)

Scrub Typhus

Typhus also appears to have been restricted to Melanesia. On his second voyage Mendana made landfall at Graciosa Bay, Ndende Island, which he called Santa Cruz, in the Philippines. The crew were struck by an epidemic with high mortality rate, which was probably typhus. The illness lasted more than 15 days and the crew were delirious with a high fever. (Miles 1997, p 22) Typhus mortality is from 1 to 60%; however, the Ndende strain is very virulent. (Miles 1997, p 25 - 6) Typhus is caused by bacteria of the Rickettsial species and can be transmitted by a number of

arthropods. According to Diamond, typhus microbes developed a new route of transmission in humans via lice. (Diamond 1998, p 209) There are several strains of typhus; however, Miles is referring specifically to scrub typhus of which, the trombiculid mite is the vector and Asian rats, *Rattus exulans*, are the sylvatic reservoir of *Rickettsia tsutsugamushi* in the Pacific. The introduced *Rattus rattus* and *Rattus Norvegicus* have also become vectors at Ndende indicating that Ndende may be the initial source for the Pacific. The rat *Rattus exulans* spread throughout the Pacific with the migration of humans. However, the vector, the trombiculid mite, does not appear to have spread beyond Vanuatu. (Miles 1997, p 25 – 6) While it is clear that typhus did not establish outside of Melanesia, the possibility remains that the occasional rat travelling on board ships from Ndende into the Pacific may have caused small pockets of disease, however, an enzootic focus has not been established.

Filariasis

Elephantiasis, more correctly known as filariasis, is endemic to Melanesia and some regions of Western Polynesia. The causative parasite in elephantiasis is *Wuchereria bancrofti*. In Melanesia filariasis is generally periodic and microfilaria are numerous in the blood at night. This is apparently due to the night biting mosquito vector *Culex quinquefasciatus*. On Cook's second voyage he recorded swollen ulcerated legs, feet and testicles were common in men at New Caledonia, (Howe 1984, p 48) and Ha'apai, Tonga. (Howe 1984, p 48) There is also early evidence of filariasis in Samoa. (Miles 1997, p 71 - 2) To the east and into Fiji the filariasis parasite has adapted to a day biting mosquito but is less common. (Miles 1997, 72 - 3) Filariasis appears in the pre-contact Cook Islands but appears to be absent from Rapanui and from Kiribati until 1841. (Miles 1997, p 73) The existing evidence suggests that filariasis was endemic in areas of Western Polynesia when Europeans arrived in the Pacific, but has since spread into some regions of Eastern Polynesia. Although filariasis managed to establish in some eastern Polynesia regions there is no evidence for filariasis in New Zealand as there were no suitable vectors and the climate in New Zealand is unsuitable. (Miles 1997, p 73) From this evidence it can therefore be established that no arthropod diseases would have travelled to New Zealand with the initial immigrants.

ACUTE INFECTIOUS EPIDEMIC DISEASES

Acute infectious diseases have a relatively short incubation period, a relatively short infectious period and require a reasonable population to sustain them. Pacific voyages would have been arduous and anyone suffering a serious illness or acute infection may not have survived the journey. (Miles 1997, p 12) In addition, the conditions for survival of acute infectious diseases upon arrival at any given location are not clearly established. (Miles 1997, p 14) Many of the Pacific Islands would not have had the population or a sufficient birth rate of naïve suspects for many acute infectious diseases to become endemic. For instance, for measles to become endemic in a population it would require a population base of 500,000 and a naïve suspect of 15,000 per annum. (Miles 1997, p 14) Therefore, if the diseases had survived the long journey they would probably have died out due to the population size.

The Origins of Acute Epidemic Infectious Diseases

As discussed above, many acute infectious diseases could not have originated in the Polynesia as many of the epidemic diseases that have swept through human populations originate from animal diseases and are therefore known as zoonoses. Smallpox, influenza, tuberculosis, malaria, plague, measles and cholera have all evolved from diseases of animals to become human diseases. (Diamond 1998, p 196) These infectious diseases fall into two categories: those that have jumped species and are now transmissible human to human diseases and those that have not fully jumped species and are still transmissible from animals to humans. Some of the diseases that have jumped species include: tuberculosis from cows, smallpox from cows and other species that have pox diseases, influenza from pigs and ducks, pertussis (whooping cough) from pigs and dogs, falciparum malaria from birds, and measles from cattle (rinderpest). (Diamond 1998, p 207) However, Miles believes the measles virus is related to rinderpest in cattle and canine distemper in dogs and postulates that the three viruses may have a common ancestor. (Miles 1997, p 16) Effectively, contact between the three species: human, cattle and canine may have created a new

pathogen.⁵ A number of diseases can still be contracted from animals but have not jumped species to become human diseases. These include leptospirosis from dogs, psittacosis from chickens and parrots, brucellosis from cattle and tularaemia from wild rabbits. (Diamond 1998, p 207) With the exception of birds and ducks, none of these animals existed in the Polynesia prior to the arrival of human beings. There was therefore no reservoir from which these diseases could develop. From this alone it could be argued that these diseases did not exist in pre-historic Polynesia and therefore did not exist in New Zealand.

DISEASES INTRODUCED INTO POLYNESIA BY EUROPEANS

There are many diseases that were introduced into Polynesia and therefore into New Zealand, such as Hepatitis A, Enterobacteriaceae, ringworm, leprosy, tuberculosis and venereal diseases and a cluster of lesser ailments, such as ulcers and eye diseases that are more likely to be symptoms of venereal diseases or tuberculosis.

Hepatitis A

Hepatitis A was probably introduced into the Pacific by Europeans. (Miles 1997, p 95) In 1778 Cook's crew developed yellow jaundice while travelling between Tahiti, Tuamotu and Hawaii. (Miles 1997, p 95) Miles believes this was hepatitis A, which has an incubation period of three to four weeks. He believes the infection may have occurred at Tuamotu and incubated on the journey. (Miles 1997, p 95) In 1821 yellow fever was reported at Hawaii, however, the mosquito vector is not established there and the disease was probably not yellow fever but hepatitis A. (Miles 1997, p 96) Given the low incidence and sporadic evidence hepatitis A probably did not establish widely in Polynesia.

Enterobacteriaceae

Broadly speaking gastro intestinal diseases were introduced into the Pacific. This group of disease causing micro-organisms includes plant and animal pathogens. Salmonella infections from vertebrate animals were probably not common in the Pacific due to a lack of hosts; they can establish chronic infections in the host, but not usually in humans. (Miles 1997, p 91) In addition, there have been far fewer sero-

⁵ A pathogen is an agent capable of causing disease.

types recorded in New Zealand than there have been in many small European countries, (Miles 1997, p 91) suggesting a late establishment and diversification of strains. The exception is *Salmonella typhi*, also known as enteric fever or typhoid, and paratyphoids. These diseases do establish chronic carrier states in humans and can survive in small populations. However, there is no evidence of pre European existence. (Miles 1997, p 91) There are a number of lesser diseases in this class that all appear to have been introduced, such as shigellae and amoebic dysentery. Miles suggests that shigellae are not likely to have established in small isolated populations, as the disease is not a chronic carrier. (Miles 1997, p 90) Amoebic dysentery can establish a chronic state, but is not likely to have been pre European. (Miles 1997, p 91) In the 1880s Fijians claimed that it was introduced by Europeans, and it was previously very uncommon in Vanuatu. (Miles 1997, p 90) while this is a large genus with many species, the evidence suggests that of the gastro-intestinal diseases studied most were introduced into Polynesia and therefore into New Zealand.

Ringworm

Ringworm was introduced into some regions of western Polynesia, the Friendly Islands (Cook Islands) and the Society Islands (Tahitian Islands) after the arrival of Europeans. In 1768 Bougainville recorded leprosy at Vanuatu, however, Miles believes this was probably *Trychophyton concentricum* a fungal organism of *Tinea imbricata*, a form of ringworm. (Miles 1997, p 38) Miles does not comment on whether he considers ringworm to be introduced or endemic, but the evidence from Western Polynesia suggests it may be introduced. On Captain Cook's second voyage the journals recorded tetter (ringworm) at Ha'apai, Tonga, (Howe 1984, p 48) (Miles 1997, p 93) and it was also reported at Tuvalu in 1841 and Tokelau in 1874. (Miles 1997, p 93) In Eastern Polynesia the Cook journals recorded ringworm, (Miles 1997, p 33 - 4) (Miles 1997, p 92) although there was no evidence of skin eruptions at Niue in 1873 or at Easter Island. (Miles 1997, p 92) Given the available data Miles concludes that there is a lack of evidence for pre-European dermatomycoses⁶ in the Pacific. (Miles 1997, p 93) The evidence that ringworm was endemic any further east than Melanesia is insufficient. It would seem ringworm spread into Western

⁶ Dermatomycoses are fungal skin infections.

Polynesia after the arrival of Europeans in the Pacific and was therefore not endemic to New Zealand.

DIFFERENTIATING VENEREAL DISEASES, TUBERCULOSIS AND LEPROSY

As early observers were frequently confused about the pathology of the symptoms of disease, it is important to trace the history of venereal diseases, tuberculosis and leprosy in the Pacific. European travellers introduced tuberculosis and venereal diseases into the Pacific very early in the period of European exploration as a result of the high incidence in European cities and the lack of knowledge of carriage or cure. Leprosy was not introduced until much later. While it is known that these diseases were introduced, early European observers often recorded symptoms as diseases, and these were often symptoms of diseases such as tuberculosis or venereal disease, rather than diseases in their own right. These symptoms included tropical ulcers and eye diseases. Anderson commented on 'Large, dangerous ulcers of a corrosive nature, often swellings, so as to be without feeling, of the arms and legs; and tumours of the testicles'. (Miles 1997, p 33 - 4) In Tahiti evidence of 'ruptures, rheumatism and body sores' were recorded. (Howe 1984, p 48 - 9) Twenty years later William Bligh, recorded 'Cancers, Consumptions, Fevers, Fits and the Scropula [sic] in a shocking degree', in Tahiti. (Howe 1984, p 49) According to Anderson inhabitants of the Friendly Islands had a corneal imperfection. From this evidence it appears a variety of diseases were present. Syphilis, leprosy and tuberculosis can all cause ulcers. Leprosy can cause loss of sensation, and gonorrhoea and tuberculosis can cause swelling of the testicles, eye diseases and rheumatism. All of the above symptoms were therefore possibly symptoms of these three diseases.

Venereal Diseases

Venereal diseases were introduced into most islands with European contact. Howe suggests that gonorrhoea and syphilis were introduced very early, probably around the 1770's. (Howe 1984, p 90) Miles also comments that there is evidence for the introduction of gonorrhoea and syphilis in the Pacific Region, (Miles 1997, p 61)

however, he claims that it is difficult to ascertain from 18th century descriptions of disease when gonorrhoea was introduced. (Miles 1997, 60 - 1)

The evidence for the introduction of syphilis is much clearer as the disease has a number of obvious symptoms, although there is some difficulty in differentiating between syphilis and yaws.⁷ Both of these diseases are caused by the species *Treponema*, a spirochetal bacteria. *Treponema pertenue* is the causative micro-organism in yaws while *Treponema palladium* is the causative agent in syphilis. Most strains of *Treponema* 'are biologically and immunologically intermediate between syphilis and yaws', (Miles 1997, p 54) therefore, they may all be products of spirochetal hybridisation. Where yaws existed syphilis did not establish. This is probably due to cross immunity, (Miles 1997, p 54) and is therefore another indicator that they are closely related micro-organisms.

Carbon dating of skeletal evidence of treponemas in the Pacific suggests the micro-organisms were introduced. While most of the skeletal remains have been carbon dated, this work was done between 1952 and 1976. There have been advances in the accuracy of carbon dating since that time that may alter the dates, however, skeletal evidence of treponemas at New Guinea is dated from 1000 to 16000AD. (Miles 1997, p 54) This would put the date as after Pacific migration, therefore, the treponemas would not have been transported across the Pacific by the early migrants. At Kiribati and Tuvalu treponemas were introduced around 1860s, (Miles 1997, p 58) and Niueans claim the diseases were introduced by Samoans in the mid-nineteenth century. (Miles 1997, p 58) Skeletons from Tongatapu showed evidence of treponemas that have been dated as pre-contact, although no date is given and skeletal evidence from the Marquesas is dated 1000 –1700AD. Treponemas were not present in New Zealand, French Polynesia or universally through the Pacific. (Miles 1997, p 58) The evidence from Melanesia and Western Polynesia suggests treponemas were introduced.

There is some question as to whether early reports of syphilis in the Pacific were yaws or syphilis. (Miles 1997, p 54) However, there are several arguments that point

⁷ It is not clear whether yaws was endemic in the Pacific.

to the disease being syphilis. First, while yaws is a common childhood disease early explorers do not observe these sores on children. Second, the crew on the ships are men not children, this would suggest that if they were carrying any treponemal disease it is more likely to be syphilis. Third, the Tahitians claimed the disease came off a ship, probably Bougainville's, several months before Cooks' arrival. Finally, the areas in which skeletal evidence of treponemas has been found are areas where there are recorded visits by Spanish sailors in the 16th century. Columbus' crew visited America in the 15th century, contracted syphilis, and took it back initially to Spain, therefore Spanish sailors were more likely to have spread the disease in the Pacific prior to the arrival of other European explorers. For these reasons, the disease was more likely to have been syphilis than yaws.

Chlamydia appears to have been endemic to Papua New Guinea but introduced into other regions of the Pacific. According to Miles urethritis and inflammation of the fallopian tubes can all be caused by the same micro-organism that causes chlamydia. (Miles 1997, 61) Miles believes chlamydia was introduced into the Pacific, with the exception of New Guinea, in the post European period. (Miles 1997, p 61) Miles does not give any suggestion as to why this disease, which does not have a sylvatic phase, did not spread eastward with the original immigrants.

Tuberculosis

Tuberculosis is a disease that has affected humans for centuries, however, the Pacific was tuberculosis free until the arrival of Europeans. Tuberculosis DNA has been isolated from a 1000-year-old Peruvian mummy. The procedure for identification did not distinguish *Mycobacterium bovis*, the causative agent for tuberculosis in cattle and humans, from *Mycobacterium tuberculosis*, the causative agent in humans, (Diamond 1998, p 212) although, both pathogens are very closely related. While tuberculosis is a human disease it can still be transmitted from cattle and other animals, however, the animals necessary for its transmission did not exist in the Pacific until Europeans introduced them. If tuberculosis were to have existed in the Pacific it would have had to exist as a human to human disease. However, Diamond does not believe that tuberculosis was a disease of the New World. (Diamond 1998, p 212) Miles, who argues that a high incidence of tuberculosis coupled with low resistance suggests

tuberculosis was not present in the pre-European period, supports this. (Miles 1997, p 63)

There is also limited archaeological evidence to suggest pre-European existence. While some samples have been dated with an early date range prior to known exploration of the Pacific by Europeans, the wide date range of the samples makes date identification difficult. There is one skeleton from Papua New Guinea that indicates pre European tuberculosis, (Miles 1997, p 69) however, the dates given by Miles of 1000 to 1600 AD fall within the period of European contact. There was no evidence for pre European tuberculosis in Samoa. (Miles 1997, p 69) Miles did not comment on other Western Polynesian groups, presumably because there were no samples. Two skeletons from the Marquesas suggest the possibility of pre European tuberculosis, (Miles 1997, p 69) however, dates are not provided. The paleopathology in Eastern Polynesia is scant to non-existent and tuberculosis doesn't appear to have existed in the pre-European period. (Miles 1997, p 69) By 1829 there is evidence of tuberculosis in the Society Islands (Tahiti), which were frequented by Europeans from 1767, however, there is no evidence of tuberculosis in the Tuamotu Islands although the earliest European contact was in the 16th century. (Miles 1997, p 64 -5) With only two samples suggesting only a possibility of tuberculosis in the Marquesas and no other archaeological evidence for its existence elsewhere in the Pacific, tuberculosis was more than likely introduced.

Leprosy

The evidence for pre-contact leprosy is tentative. (Miles 1997, 39) The use of the word leprosy was widely used by 18th century explorers and applied to a variety of diseases, which were not Hansen's disease, as we know leprosy today. (Miles 1997, p 37) Leprosy was not endemic in the South-East Pacific until the latter half of the nineteenth century. (Miles 1997, 39) There is also a lack of evidence in skeletal remains through the Pacific. (Miles 1997, p 52) There is some suggestion from eyewitness accounts of leprosy in China in the first millennium BC. (Houghton 1980, p 133) There is also a finger bone from Lau, Fiji dated to 500 BC that shows signs of erosion consistent with that of leprosy, (Houghton 1980, p 133) however, this limited evidence is not conclusive evidence for the existence of leprosy in prehistoric Pacifica. There are two other factors not addressed by Miles that cast doubt on the pre-European existence of leprosy. First, there is no evidence for Pre-European

leprosy in Melanesia. (Miles 1997, p 37) As discussed above the Polynesian people came from Melanesia and the spread of other diseases reflects migration patterns to some degree. Second, the difficulty in clinically diagnosing leprosy and differentiating the disease from other diseases with similar clinical symptoms still exists in the 21st century. (Harrop 2002, p 4) (Meltzer 2002, p 8) With the tentative evidence for its pre-European existence it seems most likely that leprosy was introduced throughout the Pacific at a later date.⁸

It is difficult to determine whether tropical ulcers were endemic. As tuberculosis and syphilis cause ulcer-like sores the early evidence for ulcers could be syphilis, scrofula or some other disease. At Ha'apai, Tonga on his second voyage Captain Cook recorded that 'disorders which affect their appearance, but not common, are large broad ulcers which appear in every part of the body' and that 'boils & blotches are often met with'. (Howe 1984, p 48) Miles believes these ulcers were also possibly present in the Cook Islands and Samoa. (Miles 1997, p 92) However, according to Miles, it is difficult to determine which micro-organisms are causative (Miles 1997, p 92) and the evidence he has collated is post European contact. Furthermore, Miles comments that tropical ulcers are prevalent in children in modern times, and sometimes adults, (Miles 1997, p 92) whereas, the early evidence does not suggest that the observations were of children. Miles offers no evidence for tropical ulcers in Melanesia or the rest of Eastern Polynesia; therefore the causative agent may not have migrated with the original migrants. Therefore, tropical ulcers may possibly be ulcers caused by diseases introduced into Polynesia.

A number of eye diseases may have been caused by venereal diseases or tuberculosis. Ophthalmia was recorded in 1839 in Samoa. The growth started from the corner of the inner eye and spread across the pupil until the grey opaque patches and the edge of the cornea joined together. Miles comments that this condition was possibly pterygium, which is believed to be caused by ultra violet light and may be exacerbated by dust, infection and other irritant factors. (Miles 1997, p 80) Miles does not describe the types of infection or whether they were from introduced diseases. However, in 1923 another

⁸ J Z Montgomerie has explored the possibility of leprosy in pre contact New Zealand in his article Leprosy in New Zealand in JPS 97 (2) pp 116 – 146, and concluded it was introduced after European contact.

Samoan eye disease was first named *Diplococcus samoensis* (Leber) but was later recognised to be *Neisseria gonorrhoeae*. (Miles 1997, p 80)

Trachoma, an eye disease, can be caused by the micro-organisms that cause chlamydia or the micro-organisms that cause gonorrhoea. (Jawetz E. Melnick J. L. & Adelberg E. A. 1970) (Miles 1997, 61) As it has already been established that both chlamydia and gonorrhoea are introduced into most of the Pacific after European contact it can be assumed that the same applies to trachoma. In addition, the evidence Miles gives for trachoma is all post 1900. (Miles 1997, p 82 - 3) He comments that trachoma *Chlamydia trachomatis* may be an exacerbating factor in cases of pterygium, (Miles 1997, p 81) as discussed above, which involves growth across the cornea.

At Tonga in 1779 Anderson the surgeon on Cook's third voyage recorded evidence of diseased corneas. (Miles 1997, p 79) Eye inflammations, extreme puffiness and purulent copious discharge were also recorded at Tonga in 1817. As there is ample evidence to suggest that both gonorrhoea and trachoma were introduced, it is reasonable to suggest that these eye diseases were due to venereal diseases, however, purulent discharge can also be caused by tuberculosis and that is another possibility to consider. Overall it would seem tropical ulcers and eye diseases were probably symptoms of introduced diseases rather than diseases in their own right.

DISEASES THAT MAY HAVE BEEN ENDEMIC

Chronic illness and latent infections may have survived a Pacific journey, and there is evidence that sub acute and chronic infections have spread between Pacific Island groups. (Miles 1997, p 14) Latent infections can survive in much smaller populations, (Miles 1997, p 15) and these diseases would have been readily transportable. Chronic, latent and sub acute diseases do not always display obvious symptoms and are not always evident to the casual observer, and may not have been reported by the early explorers. There are two viruses and some strains of two bacterial groups that may have spread across the Pacific and into New Zealand prior to European contact but the evidence is very unclear. The viruses were the causative agents for hepatitis B and herpes, and the bacterial groups were streptococci and staphylococci.

Hepatitis B

Hepatitis B may have spread across the Pacific with the initial immigrants. It is endemic in the Pacific today, although there is a higher incidence in Melanesia and Micronesia than in Polynesia. (Miles 1997, p 96) There are a number of strains of Hepatitis B; the dominant strain, which is common in South-East Asia and Japan is *adr*. This is different to the common strain in European countries, which is *adw* and *ayr*. (Miles 1997, p 96) The difference in strains would suggest that hepatitis B came into the Pacific with the original migrants although its spread has been relatively slow. While hepatitis B spread through the Pacific to some degree Miles does not identify exactly where it has spread to and it is not clear from his evidence whether hepatitis B is endemic to New Zealand. Furthermore, in 1840 reports of hepatic disease were rare and the Pacific remained exempt from biliary disease. (Miles 1997, p 95) It is therefore possible that hepatitis B was not endemic to New Zealand at that time.

Herpes Viruses

Herpes virus hominis type 1, which can cause scarring of the cornea and blindness, may have been introduced into the Pacific with the initial immigrants, (Miles 1997, p 84) although the evidence is unclear. According to Miles the herpes viruses have a long-term latency and have been associated with the kerato-conjunctivitis virus, however, Jawetz *et al* point out that kerato-conjunctivitis is not a virus but a disease caused by the herpes virus. (Jawetz E. Melnick J. L. & Adelberg E. A. 1970, p 407) Miles also suggests that epidemic kerato-conjunctivitis, which has caused epidemics since 1950, was not likely to have been present in the pre-European period. It is therefore unclear what Miles is meaning unless he is meaning there is a specific strain of herpes virus that causes kerato-conjunctivitis. The disease labial herpes simplex can also be caused by *Herpes virus hominis type 1*, which can transfer to infants. Although the initial symptoms are often minimal there may be long-term latency. However, according to Chandrosoma and Taylor, genital herpes is most often caused by *Herpes virus type 2*. (Chandrosoma P & Taylor C. R. 1995, p 870) Miles comments that there is no evidence for the existence of genital herpes in pre-European Pacifica. (Miles 1997, p 61) It does not appear that Miles is inferring that the initial immigrants introduced both viruses into the Pacific region, although

Chandrosoma & Taylor comment that there is significant cross over between the two viruses. (Chandrosoma P & Taylor C. R. 1995, p 870) Perhaps one has developed from the other. Unfortunately Miles does not set out his evidence for his assertion that *Herpes virus hominis type 1* was introduced by the original immigrants nor does he describe the spread or incidence of the virus or the related diseases. How widespread *Herpes virus hominis type 1* is not clear from the evidence. It may have spread throughout Eastern Polynesia and into New Zealand, but the evidence is very uncertain.

Streptococci

Some streptococci strains may be endemic but others were introduced. Streptococci infections in humans are more often caused by streptococci Group A. (Schleiss 2002, p 1) However, some descriptions of the diseases that can be caused by streptococci Group A are inaccurate and confuse the evidence. Miles comments that erysipelas is often mentioned in the early literature but the term erysipelas had a much wider use than it does today. In 1829 the term erysipelas was used to refer to shingles, which is caused by *Herpes zoster*. Today erysipelas refers to a dermal streptococcal infection. (Miles 1997, p 88) Therefore, descriptions of erysipelas in the early literature do not necessarily indicate streptococcal infections or diseases. However, post tattoo complications may have been group A haemolytic streptococcal infection, which are now known as erysipelas. (Miles 1997, p 88) Miles suggests that group A haemolytic streptococci was probably present in pre European New Zealand. (Miles 1997, p 89) This would suggest that group A haemolytic streptococci was probably present throughout the Pacific. Given its universality some mild strains of streptococci were probably present throughout the Pacific, but later epidemics of rheumatic fever and scarlet fever would suggest that some strains were introduced. It is reasonable to assume that the original migrants distributed some streptococcal strains.

Staphylococci

Some staphylococci strains may have been introduced. Staphylococci infections in humans are usually caused by the micro-organism *Staphylococcus aureus*. According to Herchline 20 – 30% of the American population are populated persistently with staphylococci. (Herchline 2002, p 2) However, this does not mean that all other people around the world have the same staphylococcal colonisation rate. Colonisation rates

may be higher or lower. Nor does it mean that several hundred years ago the colonisation rates were the same. Osteomyelitis is an inflammation of the bones caused by *Staphylococcus aureus*. There was no evidence of osteomyelitis in skeletal material from Papua New Guinea, Tonga and the Marquesas as the result of a study in the 1960s and 1970s. (Miles 1997, p 88) This would suggest an absence or low colonisation rate throughout the Pacific. However boils, which are also caused by staphylococci, were described at Hawaii on Cook's third journey in 1779 and in Tahiti and Tuamotu in 1829 'inflammatory tumours' were described. (Miles 1997, p 88) As the evidence for boils is post-contact and there is a lack of evidence of pre-contact osteomyelitis, it is possible that some staphylococci strains were introduced.

This overview of diseases in the Pacific region does not cover all of the potential diseases but is limited to diseases commented on or studied by experts in the field. The evidence suggests that there were a number of diseases endemic to certain regions of the Pacific but not all of these diseases were universal throughout the Pacific. Diseases such as malaria, arboviruses and typhus did not spread beyond Melanesia. Filariasis is endemic to Melanesia, Western Polynesia and some areas of Eastern Polynesia although the evidence suggests filariasis continued to spread after Europeans arrived in the Pacific. Of the diseases that could be expected to be found in island groups such as Samoa, Tonga, Hawaii and the Society Islands with significant inter island communication the establishment of filariasis was restricted to Samoa and Tonga. This supports Miles' argument that many of these diseases have difficulty in establishing enzootic focus. The evidence suggests that ringworm, leprosy, eye diseases, gonorrhoea, syphilis and other treponema, tropical ulcers, tuberculosis, chlamydia, enterobacteriaceae and hepatitis A were all introduced. Micro-organisms such as hepatitis B, herpes virus hominis type 1, staphylococci and streptococci may possibly have some endemicity. The evidence for hepatitis B and herpes virus hominis type 1 is more suggestive of endemicity than the evidence for streptococci and staphylococci; however, there may be specific strains of these cocci that were endemic. Of the micro-organisms and diseases reviewed in this chapter only these last four had any possibility of being transported to New Zealand by the initial immigrants.

CHAPTER 2: DISEASES GENETIC OR ENDEMIC TO NEW ZEALAND

Before the impact of introduced diseases can be discussed we need to have an understanding of what the state of Maori health was prior to their arrival. To ascertain the state of Maori health prior to contact archaeological records have been relied upon. Philip Houghton is the authority on skeletal evidence in New Zealand archaeology, and Houghton's findings offer the best analysis of pre contact Maori health. However, Houghton comments that there are limited archaeological samples in New Zealand. (Houghton 1980, p 78) Therefore, the findings need to be considered with some caution. To fill out the picture, other data has been gleaned from the eyewitness accounts of early European observers. Obviously, the use of post-contact evidence has its own difficulties. In some instances there are differences between these two sources, however, they are not irreconcilable. This chapter will bring forward evidence that prior to the introduction of infectious diseases by Europeans, Maori enjoyed a reasonably low incidence of disease. Genetic diseases such as cancers, kidney disease, heart disease and gout all appear in the archaeological evidence and a number of deformities are described in the eyewitness evidence, however, there are limited cases. As discussed in chapter one a number of micro-organisms may have journeyed to New Zealand with the initial immigrants, but there is little evidence for either their existence or their pathogenicity.

Genetic Disease

Many genetic diseases could have been imported into New Zealand with the founding immigrants and the larger and more diverse the founding group the greater the risk of importing a wider range of genetic diseases. While the genetic evidence suggests that a relatively small founding group colonised Polynesia, which would result in a limited gene pool, the archaeological evidence in New Zealand is suggestive of a more diverse founding group or multiple founding groups of mixed Polynesian origins. (Houghton 1980, p 80) For example, there are very slight physical and linguistic differences between Maori of the North Island East Coast and the Central North Island, in comparison to Maori in other regions, (Houghton 1980, p 78) suggesting different founding groups. In addition, there are slight indications of earlier settlement in the South Island, although the evidence is not conclusive. (Anderson

1989, p 9) In addition, studies of artefacts found in New Zealand sites also suggest multiple origins, such as the Marquesas, the Cook Islands and the Society Islands. Therefore, multiple Polynesian origins may be a possibility.⁹ However, given that the gene pool into Polynesia was relatively limited, the gene pool in New Zealand would have similar restrictions.

The genetic diseases identified by archaeological research, which indicates that gout, kidney stones, hardening of the arteries and cancer affected pre-historic Maori. Modern Maori have a high incidence of gout that differs slightly to gout seen in Europeans. Gout is a metabolic disease that occurs when the body is unable to cope with oxalate in the diet. Calcium oxalate crystals are deposited in soft tissue such as the earlobe or kidneys; the classic deposits for gout are in the big toe. There is one example of possible prehistoric gout and one incident of kidney stones, probably as a result of recurrent kidney infections associated with pregnancy. (Houghton 1980, p 140 - 141) Houghton does not explain whether the inability to cope with oxalate in the diet is due to a genetic propensity amongst Polynesians, or whether the diet in New Zealand was too high in oxalates for human consumption. However, there is also prehistoric evidence of hardening of the arteries (atherosclerosis) from a Chatham Islander aged about 50 and a similar sample was also discovered in Tonga, although the sample age was lower. (Houghton 1980, p 143) As atherosclerosis is also caused by a build-up of calcium in the arteries, there may possibly be some genetic propensity. However, this evidence is very tentative.

Two cases suggestive of cancer have been found. One, dated from 1750AD is in the spine of a girl aged about 11 years. (Houghton 1980, p 144) Houghton comments that the disease appears to be adrenal cancer, which occurs in very young children. Although the girl is a little too old for this form of cancer, it is the most likely cause. (Houghton 1980, p 144) The other is in the cranium from a young woman aged about 20 years old, at the Wairau Bar. The cranium has erosions inside the skull suggesting a secondary cancer or histiocytosis – X. (Houghton 1980, p 144)

⁹ The suggestion that Maori originate from more than one source is not an attempt to resurrect the 'Great Fleet' theory, which has been disproved, however, these indicators of multiple settlement must be considered as they may have an impact on health.

Birth defects and deformities in pre-contact New Zealand have not been identified from archaeological samples. Certain age groups are archeologically more difficult to study than others. There are inherent difficulties in such a project. The bones of infants and children are very soft and decay over time. Therefore they may no longer exist or they may be too fragile to be studied. (Houghton 1980, p 95 - 98) ¹⁰ However, although the archaeological evidence mentions bone fractures and breaks, there is no mention of birth defects and deformities. There are two possible explanations for this. First, there may have been a very low incidence of birth defects and deformities in pre-contact Maori society or second, the practice of infanticide reduced the incidence of deformities. While the archaeological evidence does not offer any evidence for birth defects or deformities, or give any explanation as to the lack of evidence, eyewitness accounts describe a number of deformities. However, it is sometimes difficult to ascertain whether they are due to injury rather than genetic disorders. The first observation of deformity was in 1814 when J L Nicholas recorded one instance of a man who had a hunched back and crooked legs. (Nicholas 1817, p 102) According to Nicholas the man had been born this way and was the only case of deformity he saw while in the country. In 1820 Captain Cruise described a young boy who had a distorted hand and arm that he attempted to hide under a mat. Cruise did not explain whether the injury was an accident or a birth defect. (Cruise 1957, p 98) This is the only recorded case, in the literature reviewed, of a deformity in a child. Ensign McRae also commented that he saw a person with a deformity but he didn't describe it.¹¹ (McNab 1908, p 540) As an example, in 1838 Joel Polack commented that deformities were rare (Polack 1838, p 273) as were imperfections in infants. (Polack 1838, p 273) He did comment that a chief at Mangakahia had deformities, (Polack 1838, p 273) however, he did not describe them. He also saw one man, a dwarf with his knees and feet inverted. (Polack 1838, p 273) Polack summed up by saying that of the thousands of Maori he had met these were the only deformities he had seen. (Polack 1838, p 273) All of the above cases were recorded at the Bay of Islands. Dr Dieffenbach recorded cases of club-foot and hair lip, (Dieffenbach 1843, p 22 - 23) during his travels in Cook Strait and the lower North Island, however he did not describe how many such cases he observed. Some individuals, such as Te

¹⁰ The age group of 16 to 26 is the best age for study due to the developmental changes in bone and skeletal structures. The outer limit of bone changes is about 30.

Rauparaha, had six toes and Dieffenbach commented that there were several members of one family with an extra digit. (Dieffenbach 1843, p 22 - 23) At Waikouaiti, Tuhawaiki's fourteen-year-old son, Kehau, had six toes on each foot, as did his grandfather Te Whakatupunga. (Shortland 1974, p 82) (Richards 1995, p 123) According to Shortland having six toes on each foot was considered a sign of greatness.¹² Throughout the observational literature there are no recorded instances of deafness and Edwardson specifically stated deafness was unknown at the South Cape. (McNab 1909, p 325) Cases of blindness were attributable to disease or injury.

Endemic Diseases

While the small sample of skeletal remains in New Zealand has not offered any secure conclusions as to the period of Maori settlement of New Zealand Diamond dates the colonisation of New Zealand at around 1000 AD. (Diamond 1998, p 341) This date tells us that New Zealand was settled late in the period of Pacific colonisation but before the arrival of Europeans and new infectious diseases in the Pacific. Therefore, no diseases introduced into the Pacific by Europeans were introduced into New Zealand with the initial immigrants.

Whether the micro-organisms for hepatitis B, herpes, and strains of staphylococci and streptococci, which can be pathogenic, were introduced into New Zealand is, as discussed in chapter one, uncertain. Assuming that these micro-organisms did survive the journey to New Zealand and establish here, the questions still remains whether they would have caused any significant disease. All of these micro-organisms are relatively stable, unlike the causative micro-organisms for influenza or H.I.V., which have a tendency to mutate rapidly. As a result of this stability it is possible that a symbiotic relationship had developed and therefore disease would only occur occasionally. Miles does not describe any incidence of disease caused by these micro-organisms in the Pacific prior to colonisation; it is therefore possible that they did not cause significant disease in New Zealand, if they did arrive at all.

¹¹ Cruise and McRae were together on the *Dromedary* and they both often made the similar observations, it is therefore possible that they are both describing the same case.

¹² The idea that six toes was a sign of greatness possibly arose because of Te Rauparaha's infamy.

The first European explorers in New Zealand observed no incidence of infectious disease in Maori communities; however, after their first contact with Maori at Cook Strait some crew members on the *Endeavour* became ill. Joseph Banks recorded that after leaving Queen Charlotte Sound and travelling south along the New Zealand coast-line, some of the crew were affected with an illness that caused nausea, vomiting and headache. (Beaglehole 1962, p 475) On the second voyage George Forster also recorded that members of the crew were affected by a nauseous disease after sexual connections with Maori women. (Forster 1777, p 237) As the disease was not more clearly described it is difficult to be certain that it was an infectious disease, which they had caught from Maori women. The crew were also eating fish and shellfish gathered from the harbour. These symptoms could describe, or partially describe, a number of infectious diseases, including hepatitis B, which can be spread by sexual contact. However, the symptoms could also describe poisoning from toxins in shellfish. On Cook's first voyage a number of men became ill after eating mussels and there may have been micro-organisms in the shellfish, such as listeria, which are capable of causing toxin-mediated disease. In effect, no conclusion can be drawn that the illness was caused by an infectious micro-organisms contracted by human contact, however, the possibility cannot be ruled out.

Houghton suggests that pneumonia was probably a major killer, (Houghton 1980, p 148) but that would depend on whether the causative agents of pneumonia were present in prehistoric New Zealand. According to Sharma, 'Pneumonia is defined as inflammation and consolidation of the lung tissue due to an infectious agent', typically *Streptococcus pneumoniae*. *Streptococcus pneumoniae* is carried by approximately 50% of the population without any incidence of disease. (Sharma 2002, p 2) Other typical causative micro-organisms are of staphylococcal genus. As discussed above the evidence for the transportation of these bacteria to New Zealand is inconclusive. There is a possibility that some strains of these bacteria did arrive in New Zealand, but the evidence of later epidemics suggest that many strains were not transported. Another class of pneumonia causing bacteria is the *Haemophilus* group. Miles did not present any evidence to suggest *Haemophilus* existed in the Pacific, prior to colonisation. Atypical species of causative micro-organisms include *Legionella*, *Mycoplasma* and *Chlamydia*. A range of other bacterial infections can also induce pneumonia but all of these bacteria fall into the category of introduced

species. Viruses can also cause pneumonia (Kuhn 2001), however, most of these viruses also fall into the category of introduced micro-organisms. It is therefore possible that pneumonia was not a major killer. There is no specific evidence to prove or disprove its existence.

Overall, there is insufficient evidence to argue that any of the infectious diseases discussed in chapter one were present in New Zealand prior to the arrival of Europeans, even though a number of potentially pathogenic micro-organisms could possibly have travelled with the initial immigrants. However, there are also other ways of measuring good health. If there were a low incidence of genetic and endemic diseases Maori would have been reasonably healthy. When the body's energy resources are diverted into supporting the immune system to fight infections over a sustained period, this can impact on overall general health, especially in children. When a child's development is affected by continual illness, a failure to develop to full potential can lead to shorter stature, poor teeth and difficulty healing from injuries. These factors are exacerbated by poor nutrition. The archaeological evidence for Maori shows a low incidence of Harris lines.¹³ This would indicate a generally healthy childhood and diet in those who survived to adulthood. (Houghton 1980, p 114)

When an adult has suffered these challenges throughout their life, an early death would not be uncommon. Therefore, factors such as physical wellbeing and stature, healing ability, teeth, and longevity are all indicators of the general state of health of any given group of people. In 17th and 18th century Europeans the state of health of those dwelling in the cities was considerably affected by poor nutrition - as a result of poverty in many cases. Poor sanitation and overcrowding would have created a paradise for pathogenic micro-organisms. Therefore, the immune systems of European city dwellers would have been under frequent assault and early death, especially in children, would be likely to occur. This was not the case for Maori, who lived in small villages, spent a great deal of time outdoors and had far fewer micro-organisms to contend with.

¹³ Harris lines indicate that there have been periods of malnutrition in an individual's life, especially through childhood.

From the early English and French explorers such as Cook and De Surville, came accounts of the good health of Maori. They were described as having a good physique, healthy appearance and good stature. (Hanham 2001) The eyewitness accounts and the archaeological evidence of Houghton's agree that Maori were tall, with the average height for men being 5' 8" to 6' and the average height for women being around 5' 4". (Houghton 1980, p 25, 31) (Savage 1939, p 24 - 26) Joseph Banks commented that the average height of Maori was similar to the height of the taller Europeans. (Beaglehole 1962, 11) In 1814 Nicholas commented that Maori were 'remarkably healthy people' (Nicholas 1908, p 7) and Watkins made similar comments in 1834. (Watkins 1908, p 13) In his 1821 report Commissioner Bigge's described Maori as robust and healthy people. (McNab 1908, p 590) However, Furneaux commented that Maori did not stand upright as a result of their low-built dwellings, (Beaglehole 1961, p 738) and M de Sainson on the *Astrolabe* in 1827 commented that Maori had slightly bent knees from crouching, but otherwise possessed a good physique and were tall and proud. (Wright 1950, p 206)

According to the observational evidence Maori had healthy skin and rapid healing ability, an indicator of a healthy immune system and a low incidence of skin pathogens. As discussed above there was only a limited number of potential disease causing micro-organisms that possibly arrived in New Zealand with the original migrants. One of those, hepatitis B has been discussed in relation to an illness caught by Europeans in New Zealand. The others, the herpes virus and some strains of streptococci and staphylococci are some of the more commonly known skin pathogens. Diseases such as impetigo, carbuncle, erysipelas, cellulitis, necrotising fasciitis, cellulitis, and coldsores are caused by these micro-organisms. (Chandrosoma P & Taylor C. R. 1995, p 866 - 868) However, there is no mention of these diseases or their symptoms in the earliest eyewitness evidence. Evidence of skin diseases does begin to appear in the later observations, but the possibility that they have been introduced by this time cannot be ruled out. Dieffenbach queried whether the beautiful skin of Maori might be due to the use of red ochre and shark oil combined with frequent bathing and open-air exposure. He noted that Polynesians have physiologically better skin than Europeans because of a 'greater development of the vascular papillae between the epidermis and the cutis'. (Dieffenbach 1843, p 160)

With the low incidence of micro-organisms and reasonably healthy immune systems the healing of wounds in Maori would have been relatively rapid. The risk of infection from pathogenic micro-organisms, such as strains of streptococci or staphylococci would have been very low and this would have resulted in a faster healing of wounds. While the risk of infection may have been lower the inflammation of wounds would have still occurred, as infection and inflammation are not the same thing. Inflammation occurs when there has been trauma, for instance to the skin. The immune system mounts a response to protect the site against infection and to assist the healing process. Inflammation can be characterised by redness, swelling, heat and pain. (Chandrosoma P & Taylor C. R. 1995, p 33 - 34) When a puncture-wound occurs the immune system attempts to protect against infection. Infection is when micro-organisms enter a host, perhaps through a puncture wound, and establish a colony. This can result in disease. When infection occurs inflammation increases. While there are accounts of inflammation in the eyewitness evidence there are no accounts of infection. The accounts indicate that inflammation was not particularly severe in most cases and this would indicate that there was a low incidence of infection in wounds. For example, Dr Bennett noted that Maori had excellent constitutions, recovered quickly from serious injuries, (Bennett 1860, p 405) and did not suffer from inflammation in wounds as their European counterparts did. (Bennett 1883, p 2) Bennett also commented that the inflammatory stage of a fracture was generally mild in Maori. (Bennett 1860, p 405) Whether Bennett meant inflammation or infection is not absolutely clear. However, according to Chandrasoma and Taylor, John Hunter first described inflammation in the 18th century, therefore, Bennett, being medically trained would probably have known the difference. (Chandrosoma P & Taylor C. R. 1995, p 33 - 34)

Tattooing was usually done over several years, with small areas being tattooed at each sitting. When large areas were tattooed in one sitting there was a high risk of death. This may be as a result of infection, however, if there was significant inflammation, as appears to have been the case in some instances, death could occur from an intense immune system reaction. This type of reaction is known as a stress response and death occurs when the body's defence mechanisms are overwhelmed.¹⁴ For instance,

¹⁴ These stress responses were systematically studied by Hans Selye (pronounced Shaya) in the 1950s.

Buller described how one man's face was tattooed completely in one sitting and consequently he died. (Buller 1878, p 171) Dr Savage commented in 1805 that inflammation occurred after tattooing. The patient sometimes became feverish and death could occasionally occur. (Savage 1939, p 52) Tattooing in sensitive areas such as the eyes and lips was also done in several sittings as it led to inflammation and fever and caused intense swelling and pain. (Ollivier 1986, p 160) John Rutherford, a Pakeha Maori, described his tattooing and commented that immediately after he was completely blinded, but within three days the swelling was reduced enough for his sight to be recovering, although full recovery took 6 weeks. (Craik 1830, p 136 - 137) However, according to Polack that moko or tattoo wounds on Maori healed in half the time the wounds healed on Europeans, (Polack 1838, p 271) (Polack 1976, p 97) suggesting a robust constitution.

The robustness of the Maori constitution was also observed in healing from other wounds. For example, Dr Marshall observed that Maori had a remarkable constitution and recovered from wounds that would have killed a European. (Marshall 1834, p 186) both the Reverend Butler and Joel Polack observed that Maori flesh wounds healed very quickly. (Barton 1927, p 250) (Polack 1838, p 271) (Polack 1976, p 97) Henry Williams described one old man who had been shot through the eye years before. He was still fit and well despite the injury he had sustained. (Carleton 1948, p 216) Dr Bennett also described an eye injury that destroyed the eye, although the wound itself had healed within a very short space of time. (Bennett 1883, p 4) Overall, it would appear that Maori had generally sound healthy constitutions, with a low risk of infection, although inflammation with a strong immune system response could cause death in some instances.

Teeth

According to the archaeological evidence, after 1500 AD there was a significant change in tooth wear patterns across New Zealand. The wear on teeth was rapid and the enamel had worn through to the dentine in some 12 year-olds. (Houghton 1980, p 121) In some cases tooth wear was severe to the point of exposure of the pulp cavity. At this point infection can set in and the tooth subsequently falls out resulting in a loss of molars by the age of 25. By the age of 40 most of the useful molars are gone. (Houghton 1980, p 123) Houghton does not describe which micro-organisms would

have caused the infection, however, the most likely microbe is *streptococci mutans*. (Chandrosoma P & Taylor C. R. 1995, p 464) There is skeletal evidence of erosion under teeth by infection; therefore this streptococcal strain may be endemic.¹⁵ Houghton suggests the probable cause for the change in tooth wear was a change in diet as a result of climatic change. (Houghton 1980, p 123) However, there are limited samples for the post-1700 period. There is a difference in evidence between archaeological evidence and the eye-witness accounts of the post-contact period, nevertheless continuity is provided by reports of significant wear on teeth over time as a result of a coarse diet. Edward Markham who was at the Bay of Islands in 1827 saw old people with their front teeth worn down to the gums from eating fern root. (Markham 1963, p 38) Joseph Banks had also commented that he saw old, grey-haired people with worn teeth when in Queen Charlotte Sound. (Beaglehole 1962, 21) Nevertheless, most observers noted that Maori had good teeth. Alexandre D'Ariveau on the Recherche in 1793 had remarked that Maori in North Cape had good strong teeth. (Ollivier 1986, p 31) In Northland Colenso commented that Maori had beautiful white teeth. (Gluckman 1976, p 144) The teeth of Maori in the South Island were so highly regarded that they were extracted to send back to London to make dentures. An account from the late 1830s recorded that on a visit to Putaringamotu (Dean's Bush), Maynard caught a man from the company of Wills & Sons of Regent Street, dentists, in the process of extracting the canines and incisors from the upper jaw of a dozen Maori; it was believed that no other human being had teeth as white and strong as Maori. (Dumas 1937, p 341) However, from Cook's first voyage there appears to have some evidence of dietary difference in some regions such as Poverty Bay where the teeth of the inhabitants were not well formed or a good colour. (Beaglehole 1955, p 572) Colenso also commented in the 1830s that those who lived in the Taupo and Rotorua districts had badly stained teeth due to the sulphurous water in the region. (Gluckman 1976, p 144) Overall, Maori probably had good strong teeth that were eventually affected by dietary factors such as sulphurous water and coarse fern-root. However, the initial good state of their teeth would indicate an adequate diet and reasonable health.

¹⁵ Streptococci mutans endemicity could possibly be established by DNA sampling of the skeletal evidence.

Longevity

While it would be unreasonable to expect that Maori lived to an age that far surpassed the average age of prehistoric people in other regions of the world, the fact that many Europeans commented on the number of old people would indicate that they were surprised at their longevity. This is not supported by the archaeological evidence, however, there is some possibility that the archaeological evidence may not provide an accurate assessment of longevity. The primary difficulty is that the majority of Houghton's evidence is from the Wairau Bar site. This site dates from 1300 – 1400AD, which is too early for this study. The average age of the adults studied at Wairau bar was 28 for males and 29 for females, (Houghton 1980, p 98) and Houghton claims not many individuals could be aged much over the age of 60. (Houghton 1980, p 98) However, Houghton also comments that around 1500AD climatic change led to a change in diet, (Houghton 1980, p 124 - 7) which may have led to slightly increased longevity. As there is a lack of skeletal samples for the mid to late 18th century when the first European observers arrived in New Zealand there is a lack of comparative archaeological data for the contact period. However, Houghton's estimate of not much over sixty is not that far removed from eyewitness accounts of around seventy years of age, if an allowance is made for conservatism on the part of scientific analysis and exaggeration or generalisation on the part of eyewitnesses. For example, Polack commented that a few lived to a very old age. (Polack 1838, p 272) He claimed a number of old persons attained the age of 70 years, (Polack 1976, p 126) although only one or two were aged between 70 and 80, (Polack 1838, p 153 - 158) whereas, Savage commented that some Maori appeared old before their time and others seemed to retain their youthfulness to a good age. He could not determine specific ages. (Savage 1939, p 24 - 26) Nicholas commented that Tara of Kororareka and his wife could not be much less than 70 years of age, (Nicholas 1817, p 208) (McNab 1908, p 346 - 348) and during his visit to the Bay of Islands in 1814 Nicholas claimed he saw many instances of longevity. (Nicholas 1817, p 38) However, in giving evidence to the House of Lords in 1838, he commented that he saw some old people but not many. (Nicholas 1908, p 7) It is therefore difficult to accept eyewitness evidence at face value, nonetheless, there are sufficient eyewitnesses making similar statements to accept that there were a number of old people, aged perhaps between 60 and 70 years of age.

The post contact period also offers Maori memory as a way to measure longevity. For instance, in 1821 Ensign McRae commented that he saw several people old enough to recall Cook's visit when they were young men and women. (McNab 1908, p 540) As Maori were young men and women by around the age of 12 – 14 years and Cook arrived in the Bay of Islands over fifty years before McRae, this would put these people in their early sixties. In 1814 Nicholas also met Pene of Paroa who remembered Captain Cook from his boyhood. (Nicholas 1817, p 265 - 266) This would put Pene as at least 50 years old. Hongi Hika told Rene Lesson in 1824 that he was born the year d'Fresne was murdered, (Ollivier 1986, p 146) this would make him 56 when he died in 1828. Earle was in the Bay of Islands in 1827 when he met an old man who claimed he was one of a group who attacked Furneaux in 1774. (Earle 1909, p 24) Earle also conversed with a man who was variously called King George, Shulitea or Whareumu. King George recalled that when he was a young boy and his mother Turero, who was still alive in 1827, was a young woman, Captain Cook had come to the Bay of Islands. He recalled that by the time that d'Fresne was killed he was a young man and had participated in the attack. (Earle 1909, p 122) All of these events occurred over fifty-five years before Earle's visit. Tohitapu was a chief and Tohunga of the Roroa tribe at Haumi. He claimed to have feasted on the body of d'Fresne in 1772. (Rogers 1961, p 46) In November 1833 at Thames Henry Williams met Te Horeta te Taniwha. (Rogers 1961, p 343) Te Horeta claimed he met Captain Cook when he was a boy. At Tolaga Bay in 1838 Polack met some old chiefs who had been boys when the *Resolution* had called there. (Polack 1838, p 120) All of these people would therefore have been over the age of sixty, if their recollections were true. When Tohitapu died in 1833, he would have been at least 70. Turero would have been in her 70s and Te Horeta must have been close to 70, if not older. However, the evidence from the South Island suggests that people attained an even greater age than people in the north and this can be supported with later evidence. According to Edwardson, in 1822 men at South Cape lived to be about eighty and women lived to be about eighty-five to ninety. (McNab 1909, p 263) While Edwardson's estimations may be a little high, the above anecdotal evidence does suggest that at the time of first contact Maori were living to the age of at least sixty, in some cases. The discrepancy between the archaeological evidence and the eyewitness evidence is not huge and allowing a margin of five years either way the difference can

be reconciled. It is therefore reasonable to suggest that a number of Maori survived past the age of sixty and up to seventy years of age. This would suggest reasonable health throughout their lifetimes.

Micro-organisms capable of causing disease in human-beings live, for the most part on or in human-beings. They exist for only short periods of time, in most instances, outside of the human body. Therefore, pathogenic micro-organisms in New Zealand would have arrived either with the initial immigrants or with European explorers. With the possibility of small founding groups, the possibility that any pathogenic micro-organism did not survive due to the small population is quite likely. The evidence for their existence in New Zealand at the time of European contact is in most cases slim. There is slight evidence for the existence of hepatitis B in Queen Charlotte Sound, although it does not appear to have been causing disease amongst Maori. It is possible that a strain of streptococcal bacteria was present, as indicated by the archaeological evidence. However, overall there was a low incidence of infectious diseases. Genetic diseases appear in the archaeological evidence and are more likely than infectious micro-organisms to have been the primary cause of death and disease. Overall, Maori had good physique, tall stature, good teeth, generally excellent healing ability and lived to a reasonable age indicating a healthy people who lived in isolation from disease causing micro-organisms.

CHAPTER 3: THE IMPACT OF ACUTE, INFECTIOUS INTRODUCED DISEASES

With increasing contact with the outside world Maori society, especially at the Bay of Islands, was exposed more and more frequently to infectious diseases. Once the penal colonies had become established in Australia and Norfolk Island convict vessels with overcrowded quarters became efficient transporters of diseases such as typhus, typhoid and dysentery. These diseases then spread across the Tasman to New Zealand on visiting ships. Epidemics of catarrh and influenza occurred in 1820, 1827 and 1836 - 1838 in various ports around the country and as the Australian colonies became more established and the child population grew, childhood diseases such as whooping cough, measles, mumps and polio crossed the Tasman into New Zealand.

Epidemics prior to 1800 were not recorded at the time of their arrival. Although fragmentary evidence of their existence still remained in oral history, they were recorded many years after the event. However, Europeans who visited New Zealand made observations about illnesses that they unwittingly brought with them, although there is often difficulty in identifying some of the diseases, as the descriptions are vague and incomplete. Sometimes the only way to identify the epidemics in New Zealand is by comparing them with what epidemics had occurred in Australia, the usual departure port for New Zealand at the time. While not all ships came directly from Australia, most stopped off to load, unload and re-supply. Australia is therefore a good indicator for diseases circulating in the region.

The bulk of the data in this chapter is relevant to the Bay of Islands and is not necessarily applicable to other regions of New Zealand, unless specifically stated. There are a number of factors that set the Bay of Islands apart from the rest of New Zealand during the later pre-Treaty period. First, the region became a resort for long-at-sea whalers who arrived to spend a few weeks resting and relaxing, or more correctly, in many cases, drinking and womanising. This occurred most especially at Kororareka, although other areas such as Te Puna were also affected. The region had also undergone significant change as a result of Hongi Hika's wars in the 1820s. As a result of these wars a large slave population became established at the Bay of Islands. Therefore, the Bay of Islands ceased to be typical of Maori society in many ways

from an early period. According to Maning, the slave population was in poor health. (Calder 2001, p 185 - 186) A significant slave population would have had an influence on the impact of introduced diseases especially if they were in a poor state of health, therefore, the impact of infectious disease at the Bay of Islands was not necessarily a reflection of the impact of infectious diseases in other regions. In addition, the Bay of Islands was frequented more often than other regions by European vessels, therefore there was a greater risk of disease

HOW INFECTIOUS EPIDEMIC DISEASES AFFECT HUMANS

The way in which humans and particular micro-organisms interact has implications for the impact epidemics can have. Infectious epidemic disease is usually an interaction between micro-organisms and, in this case, human beings. When an epidemic has occurred and the survivors have developed some immunity the impact of the next epidemic will theoretically be less significant. Therefore, to understand what impact the diseases may have had on the population it is necessary to have some understanding of how the causative micro-organisms behave. Micro-organisms cause illness in order to facilitate transmission to other hosts, therefore diarrhoea, sneezing, coughing, ulcers and sores are all modes of transmission for the micro-organisms. Human beings respond to micro-organisms in a variety of ways, such as increased fever, as many micro-organisms are heat sensitive and will die off if body heat increases. (Diamond 1998, p 200) The immune system views micro-organisms as invaders and white and other blood cells will hunt and kill them. However, micro-organisms also have qualities that are termed antigenic. In the process of combating antigens antibodies are developed which may provide immunity or resistance to the next contact. (Diamond 1998, p 200) Lifelong immunity can develop to diseases such as measles, mumps, rubella, pertussis (whooping cough) and smallpox, (Diamond 1998, p 200) although there can be individual exceptions. This significantly reduces the impact of the diseases the next time they strike the same population. However, this is not the case with all micro-organisms. Influenza can reoccur because the virus changes its antigenic structure and is no longer recognised by the body's antibodies. (Diamond 1998, p 200) Human death is an accidental by-product of the micro-organism life cycle, (Diamond 1998, p 199 - 200) and is usually not in either the micro-organism's or the human's best interest. For this reason both micro-organisms and human beings adapt to develop a more symbiotic relationship. Micro-organisms

that may have a life cycle of only a few days duration will adapt more quickly to humans than humans can to the micro-organisms, and generally adapt by reducing their virulence. This is why when a disease first breaks out within an immunologically naïve community there is often a high morbidity and mortality rate. As the human population develops resistance, the impact of the diseases subsides to some degree. As humans develop resistance to the micro-organisms the diseases become 'childhood' diseases, as children provide a new group of susceptible hosts. However, in immunologically naïve populations infectious epidemic diseases are more likely to kill adults than children. (Diamond 1998, p 204) Despite the much longer life span, human adaptation does occur. One such example is the Chinese resistance to syphilis. Unfortunately, human adaptation is not always perfect. Examples of maladaptation are sickle cell anaemia in Africans as a response to malaria, or the Tay-Sachs gene in Ashkenazi Jews as a response to tuberculosis, or cystic fibrosis in northern Europeans as a response to bacterial diarrhoea. (Diamond 1998, p 201) These maladaptations have now become genetic diseases.

Naïve human populations are more adversely affected by the arrival of new acute infectious diseases, than populations where the diseases have become endemic and immunity or resistance has developed. Where the diseases are recently introduced they may appear to attack the population with renewed virulence; in fact, the apparent virulence is more likely to be due to the lack of immunity or resistance of the humans. In populations where the disease has become endemic and the disease appears to attack with renewed virulence it is more likely to be due to the development of a new strain of the disease. This has recently occurred in India, where a mutant strain of measles has developed as a result of a measles vaccination campaign. (Mudur 2001)

Where Maori were concerned it would be expected that the initial contact with diseases such as whooping cough, measles, polio, mumps and other diseases normally categorised as childhood diseases, would initially have a dramatic impact on the population. However, since these diseases usually infer some immunity, their next introduction into the same environment would have meant the disease would have affected only those who either had not had contact with the disease before or those who failed to develop some immunity from the first contact, as discussed above. Usually those who had not had prior contact are those in the community who had

been born after the last contact. Infections of influenza do not usually infer immunity or complete immunity as the virus mutates and changes its antigenic structure; therefore, influenza epidemics can continue to sweep through the population time after time infecting the same people over and over. Therefore, the impact of influenza on Maori would have been epidemic dependent. Immunity to infections of typhus, typhoid and dysentery are less clearly defined. Some strains of typhus infer immunity; immunity to dysentery causing micro-organisms is dependent on the causative agent, and some immunity may occur after typhoid.

Due to the sporadic nature of the evidence it is difficult to assess the impact of these diseases on Maori society, in many cases. However, it can be assumed that if the diseases arrived in the country, first contact for Maori would have resulted in a fair degree of illness and a number of deaths. Recovery from these illnesses would have been dependent on each individual's ability to fight infection. In chapter two it was shown that Maori recovered quickly from wounds and this would indicate a healthy immune system. However, the immune system combats different infections in different ways and an ability to heal rapidly from wounds may not necessarily indicate the ability to recover rapidly from other infections. There are two groups in any population that are often more susceptible, children and the elderly, although as discussed above adults in their prime are more vulnerable to first contact with some infections.

TYPHUS, TYPHOID AND DYSENTERY

From as early as 1790 there were epidemics that came and went up and down the country. However, the descriptions of the epidemics in the literature prior to 1820 are so vague that the diseases cannot be identified from these sources. Furthermore, the reports on morbidity and mortality are also difficult to assess. However, it is most likely that the epidemics recorded from 1790 to 1795 were dysentery, typhus or typhoid. From accounts of illness in the Australian colonies during the period 1790 – 1800 these diseases occurred amongst the convicts, ships crews, and overseers. All of the diseases had been brought to the colony on the convict vessels from England, (Cumpston 1989) and vessels sailed the triangle of Norfolk, Australia and New Zealand. Therefore, if any disease were to have been introduced during this period it would almost certainly have been one of these three. There were few vessels in New

Zealand waters during the period. In October 1791 Captain Bunker on the *William and Ann*, a recently converted convict ship, called into Doubtless Bay. In April 1793 Lieutenant Hansen on the *Daedalus* called at Sandy Bay, kidnapped two locals and took them to Norfolk Island. They were returned to their homes in November on the *Brittannia*, the same year. In November 1794 the *Fancy* was in the Thames area after visiting Northland. Any one of these vessels could have introduced epidemics of typhus, typhoid or dysentery.

As Australia and Norfolk became more established and whaling and sealing industries developed around New Zealand, the number of vessels visiting our shores began to increase. As contact with the outside world increased, so too did the risk of epidemics. After 1800 Maori began to voyage across the Tasman to Australia, sometimes via Norfolk and this also increased the potential exposure to epidemics. Such was the case for Te Pahi. Te Pahi and his sons had travelled to Norfolk and then on to New South Wales in 1802 - 1803. (McNab 1908, p 334) On his return to New Zealand Te Pahi became dangerously ill. The only epidemic in the region at that time was an epidemic of typhus that had arrived at Port Jackson in 1802. (Cumpston 1989, p 41) In 1803, prior to his illness, Te Pahi was described as being 5 foot 11 1/2 inches with an athletic build and an expressive and commanding countenance. (Turnball 1813, 491- 502) Two years later, in 1805, Dr Savage described Te Pahi as 'far advanced in years and [he] had become paralytic'. (Savage 1939, p 51) Clearly, Te Pahi's seeming advanced age was a result of his illness. One of Te Pahi's wives was very ill and believed to be past all possibility of recovery. She was sent to an island near Te Puna with two attendants to care for her until she died. (Savage 1939, p 32) While there is no record of an epidemic in New Zealand at that time, Te Pahi's wife possibly contracted the illness from her husband. Of course, whether the illness was typhus or not cannot be proven.¹⁶ Whether an epidemic occurred in New Zealand at this time is also uncertain. However, Te Pahi's illness was obviously long term and debilitating, this points to typhus as his probable disease.

¹⁶ It is possible that Te Pahi's illness was tuberculosis. However, Te Pahi's son Matara died in 1808 after contracting tuberculosis in England. In later years one of Te Pahi's wives was certainly suffering from tuberculosis. However, it is more likely that Te Pahi was suffering typhus than tuberculosis at this time as an epidemic of typhus occurred in 1803 and Cumpston does not indicate that tuberculosis was well established in the Australian colonies, and it was not well established in New Zealand as far as we know.

Typhus can occur as an epidemic and it can continue to reoccur in the patient for weeks, months or even years after initial contact. (Hansen 2002) It has an incubation period of 6 to 21 days and symptoms of fever, chills and severe headache develop. The primary lesion or schar bite site is difficult to detect. The fever increases for a week to 40 - 41 C and a macular rash appears on the trunk, which can be transient or last for several days, although it may not always be present. In the second week the fever stays high and the patient becomes delirious, possibly with stupor and muscle twitching. According to Hansen, epidemic typhus may also involve Central Nervous System dysfunction. (Hansen 2002) By the third week the temperature lowers over several days, although death may occur at the end of the second week due to secondary infections such as pneumonia, encephalitis or circulatory failure. Mortality is from 1% to 60%. The illness is generally less severe in children than in adults. (Miles 1997, p 25 – 6)

As discussed in chapter one, scrub typhus was endemic to Melanesia but had not established enzootic foci outside of that region. Diamond has commented that typhus developed a new enzootic foci in human lice, *pediculus corporis*. Hansen and Cunha also comment that this vector causes epidemic typhus in humans. (Hansen 2002) However, Miles, in his discussion of scrub typhus endemicity in the Pacific, did not indicate that any other form of typhus had any endemicity in other Pacific regions. Therefore, if other forms of typhus were not endemic in the Pacific they would not have had endemicity in New Zealand, although linguistic studies have shown that Polynesians have a long association with kutu (lice). Therefore, typhus-bearing body lice may have been introduced.

As discussed above, it is not certain whether an epidemic occurred at this time; however, an incident in 1808 suggests that Maori were terrified of the possibility they would be plagued by the 'watch atua' (watch god) and this would suggest that they already had some knowledge of the impact of European atua (god) and their consequences, such as an epidemic. Captain Ceronci, the captain of a sealer named the *Commerce*, was in the harbour at Wangaroa in 1808 – 9. (McNab 1914, p 117) He showed his watch to Maori and they believed it was an atua. The watch accidentally dropped into the harbour and local Maori feared that the atua would

plague them. Soon after, a 'violent epidemic' killed many chiefs and others of the tribe and the deaths were attributed to the atua of white men. (Craik 1830, p 344) Once again the cause of this epidemic is not clear. A description given to Marsden on his first visit to Taiamai in 1814 indicates it may have been typhus, as there is no mention of dysentery. Maori told him of an epidemic that 'slew a great many of them'. Their tongues were foul and their whole bodies burned with heat. (Craik 1830, p 378 - 379) It is difficult to know what date this epidemic occurred. If it was around 1803 then this might confirm that an epidemic did succeed Te Pahi's illness. However, Maori fears in 1809 after the Whangaroa incident would suggest an epidemic had already occurred. In 1809, Ceronci, then a passenger on the *City of Edinburgh*, stopped off at the Bay of Islands for three months. Again by accident he dropped his watch overboard. Tara, a chief, who stood near him shrieked and wrung his hands in terror and distress, fearing that the Bay of Islands people would suffer the same fate the Wangaroa people had. (Craik 1830, p 344 - 345) (McNab 1914, p 121) Clearly, although the news of the epidemic at Wangaroa in 1808 had spread down to the Bay of Islands, the epidemic itself had not.

At Hauraki in 1820 Cruise commented that an infectious disease carried off great numbers of people several years before. (Cruise 1957, p 194) In January 1815, on his first voyage to New Zealand Marsden had travelled to Hauraki. His vessel was the only one in the region at that time, and Marsden himself did not record any epidemics prior to his arrival. As Marsden had travelled to Hauraki with a number of Maori from the Bay of Islands and some young men from the mission at Parramatta, the opportunity was there for the disease to be introduced. On the voyage to New Zealand a month before a young man had died of dysentery and it was common at Parramatta at that time. The young man had been in Marsden's care for some time and the disease was common at Parramatta at that time. Whether the vessel had others on board with the disease is unclear, however, it is possible that it was introduced into Hauraki at that time and caused a subsequent epidemic.

An outbreak of typhoid occurred in Queensland in 1832, (Cumpston 1989, p 55) and in June 1832 a number of natives were very ill and dying at Utuihu. (Rogers 1961, p 248) A typhoid epidemic also occurred in Queensland in 1832, although the month is not stated. (Cumpston 1989, p 55) Williams recorded in July 1832 that Maori at

Kororareka were inquiring about cholera¹⁷ after one man off a vessel in the harbour died in late June. (Rogers 1961, p 249) The disease appears to have spread further south, as Gordon Browne commented that Maori at Thames were in a sorry state due to war and sickness in August 1832. (Rogers 1961, p 254)

It is difficult to assess the impact of these epidemics. While the epidemic prior to 1820 at Hauraki is believed to have carried off many people, the literature does not give a clear assessment of how many were ill or how many actually died. While the incident with the watch would suggest that Tara was absolutely terrified of the possibility of another epidemic, and we can assume from that the impact must have been significant, the literature does not tell us how many were ill and how many died. The outbreak of typhoid in 1832 suggests a number of people were dying, but again how many were ill and how many died is not recorded. Overall it is difficult to assess the impact of typhus, typhoid and dysentery, but there is enough information to suggest that it may have been considerable.

INFLUENZA AND CATARRH

Epidemics of catarrh and influenza appear periodically in the literature. These epidemics of respiratory diseases appeared to have had a significant impact on Maori societies, most especially because they were not confined to one region, as the typhus, typhoid and dysentery appear to have been. The first epidemic of respiratory disease to appear in the literature occurred in 1820 after the arrival of the *HMS Dromedary* at Kororareka on 27 February 1820 under the command of R A Cruise. (Cruise 1957, p 21) The vessel had left Sydney on 15 February and from that date through until 9 June, three people on the vessel died. (Cruise 1957, p 28) Also on board the vessel were Maori who were returning from Parramatta. The sons of Perahiko and Te Kokee [sic], and the nephew of Te Torro [sic] had died at Parramatta, (Cruise 1957, p 30) (Cruise 1957, p 156) possibly from the same illness. While the vessel was at Kororareka a number of local Maori lived on board. Korokoro's child became unwell and was taken off the vessel for treatment by a tohunga (priest). It is not stated whether he survived. (Cruise 1957, p 51) Cumpston comments that an epidemic of

¹⁷ The terms cholera and typhoid appear to have been interchangeable in this period. There are no recorded outbreaks of cholera during this early period.

catarrh was present at NSW in 1820, (Cumpston 1989, p 42) and it is probably this that the *Dromedary* brought to New Zealand.

The *Dromedary* also travelled to Waitemata and Hauraki, (Cruise 1957, p 141) and then on to the Coromandel. From the Coromandel the vessel returned to the Bay of Islands. It was not until 1827 that Toupaia told Dumont D'Urville about an epidemic that had occurred in the Hauraki region in 1820. The previous chief had been Houpa, who with many of his warriors succumbed to the epidemic. The settlement of Wai Kahouranga at the mouth of the river Thames was abandoned after the epidemic and all the homes were put under tapu. The village was re-established to the south at Hauraki Bay. The *Coromandel* and the *Dromedary* travelled together to Hauraki. On board was Samuel Marsden. Hauraki Maori believed that Marsden was a powerful prophet and the epidemic was caused by the English god that he had brought amongst them. (Wright 1950, p 158)

S P Smith also records that in 1820 an epidemic spread south from Coromandel. (Smith 1910, p 268 - 9) According to Smith's informant Watene Taungatara, the epidemic was introduced by the vessel *Coromandel* when it arrived in the Hauraki Gulf in 1820. The epidemic was passed from tribe to tribe until it reached Taranaki, killing large numbers of people in its wake. At Taranaki the tohunga attempted to counter the effects of the epidemic by making a representation of a European vessel in sand, complete with mast and rigging, and chanting karakia (traditional prayers) over the vessel. Their efforts, however, were in vain. (Smith 1910, p 310 - 11) The name Te Ariki was given to the epidemic by the Taranaki people. Whether this reflects Maori belief that Marsden was responsible for bringing in the European God is not clear. The epidemic was said to be so severe that there were not enough people left to bury the dead and many thousands are said to have perished. (Smith 1910, p 310 - 11) While this figure may be exaggerated, it gives an idea of Maori perceptions of the severity of this epidemic.

An influenza epidemic occurred in several regions throughout the country in 1827 with significant impact. Dr Cunningham, a Royal Naval Surgeon in Australia, described an outbreak of influenza in 1826. (Cumpston 1989, p 43) It strongly affected the nose and eyes and was generally fatal to the old and the children. (Earle

1909, p 110) The epidemic appears to have crossed the Tasman and was affecting the inhabitants at the Bay of Islands in December 1826. (Williams 1939, p 13) Many people in the settlement were dying in December 1826 and many people were ill and some died at Taiamai. (Rogers 1961, p 31) When Earle arrived at Hokianga in 1827 the disease was already present and raging through the population. (Earle 1909, p 110) The epidemic appears to have continued through the year as Hamlin recorded an influenza epidemic in October 1827 at the Bay of Islands (Ryburn 1979, p 26) and Henry Williams recorded incidents of Maori being sick from July through to December. (Rogers 1961, p 87 - 89)

Around December 1827 *The Research* arrived at Kawakawa. Many of the people there were ill with influenza or catarrh. (Dillon 1829, p 341) No doubt the epidemic was continuing to affect villages around the region. The people of Waikari believed the influenza epidemic came off the *Research* as the vessel was in the harbour at Kororareka with a number of sick men. (Rogers 1961, p 92, 84 - 85) However, the men were suffering from an apparent attack of malaria after a visit to Manicolo and the influenza epidemic had already been in the country for several months. The local cure at the Bay of Islands for the epidemic included wrapping their heads entirely in green leaves, sweat huts and universal starvation, which according to Earle appeared to work well. (Earle 1909, p 110) Earle commented that he was amazed at their recovery from a seemingly incurable illness. (Earle 1909, p 110) However, it does appear that a number of deaths had occurred when the epidemic first struck.

The epidemic also appeared at Banks Peninsula in March – April 1827 and spread to Murihiku with a war party that had been at Akaroa. Chief Towiwi [sic] died on the return journey and chief Totoes [sic], chief at Kaika Totoes [sic], died shortly after. (Starke 1986, p 85) Boutlbee, who also succumbed to the illness, described it as a ‘severe and dangerous cold’ accompanied by a high fever and continual thirst. The illness lasted for approximately 10 – 14 days and left Boutlbee too weak to eat or walk. (Starke 1986, p 76) During the illness Ooree Pahbah [sic] and his family cared for Boutlbee.

Dumont D’Urville recorded in early 1827 that he no longer had any men sick on board the vessels, suggesting that men had been sick previously. (Wright 1950, p 143) While in New Zealand the vessel travelled to a number of ports around New Zealand

including Queen Charlotte Sound and Hauraki. There are no accounts of epidemics in these areas; however, that is possibly due to a lack of information not a lack of impact as there is little literature about these regions recorded at that time.

The Influenza Epidemics between 1836 - 1838

Influenza epidemics appear to have raged up and down the country between 1836 and 1838. Once again it is difficult to assess the impact these epidemics, or this reoccurring epidemic, had on morbidity and mortality. Nonetheless, there is evidence of sickness and death, especially by 1838.

The epidemic appears to have begun in the autumn of 1836 at Otakou, (Anderson 1998, p 193) and spread to Piraki [sic] in Akaroa. (Hemplemen 1911, p 35) The epidemic reoccurred at Akaroa in late October 1837, affecting a number of Maori. (Hemplemen 1911) In December 1837 King George and three others from Taumutu were unwell (Hemplemen 1911, p 62) and Taiaroa was sick at Piraki. (Hemplemen 1911, p 154) The epidemic continued into January 1838. (Hemplemen 1911, p 63 - 64) The epidemic also appeared in the North Island and Anne Wilson recorded an outbreak of illness at Tauranga in 1836. (Porter 1996, p 263 - 269) Several were sick with fever and headache. The disease was dubbed Puriri fever as a number of people at Puriri had had the illness. By December 1836 it had reached the Bay of Islands. (Williams 1939, p 31) When Dr S H Ford arrived in the Bay of Islands in 1837 it was still raging and killed his own children. At that time Ford had over 800 Maori patients. (Gluckman 1976, p 58)

Buller claimed that in the autumn of 1838 the epidemic claimed the lives of many old and feeble people, (Buller 1878, p 50) and then in October 1838 the *Coromandel* arrived at the Bay of Islands and another epidemic of influenza followed shortly afterwards. (Williment 1985, p 125) Dr Richard Day was on board the *Coromandel* and was able to help the sick. (Williment 1985, p 126) By December the epidemic had affected almost everyone at the Bay of Islands and many who were old, infirm or in poor health died. (Williams 1867, p 257) (Williams 1939, p 36) When the influenza epidemic reoccurred at the Bay of Islands it appears to have struck with a renewed virulence. The 1838 epidemic also appears to have affected other regions in the North Island, as in February 1838 the *Australian* under Captain W B Rhodes was at 'Kakikica' on the East Coast, apparently around Te Araroa. After setting sail

Rhodes commented that he had four crewmen sick with influenza and several more complaining of colds and sore throats. He supposed that his men had contracted this illness at the East Cape as he noticed many people there with colds and influenza. (Straubel 1954, p 80 - 82) There is evidence the epidemic was still causing illness in March 1838. (Richards 1995, p 71) (McNab 1914, p 236) Once again it is difficult to assess how many died, but it would appear that large numbers of people were affected by these respiratory epidemics. However, they were not the only diseases causing illness.

CHILDHOOD DISEASES

Mumps

The impact of childhood diseases, as they may be known to Europeans, can have a devastating impact on an immunologically naïve population such as Maori. This appears to have been the case for a number of epidemics of so-called childhood diseases that affected various regions at various times. An outbreak of what appears to have been mumps occurred in the summer of 1825 - 6. The impact on morbidity and mortality is unknown. The illness began with fever and swellings of the head and face, (Williment 1985, p 57) symptoms that would suggest mumps. Cumpston records that an epidemic of mumps occurred in New South Wales in 1824 but does not give a month. (Cumpston 1989, p 42) it is possible that this epidemic eventually made its way to New Zealand.

Whooping Cough

Another epidemic was that of whooping cough in 1828. A whooping cough like illness had occurred in Australia in March that year, (Cumpston 1989, p 42, 56) (Rogers 1961, p 145 - 147) and a number of children had died. The illness was believed to have come off the vessel *Morley*. (Cumpston 1989, 42) Within a few months it had spread across the Tasman and was sweeping through the Bay of Islands. The epidemic began in June amongst the mission children (Rogers 1961, p 135) and continued until January 1829. (Coleman 1865, p 112) Many Maori at Kerikeri and Haumi were very unwell by October (Rogers 1961, p 145 - 147) and by January 1829 many had died. (Coleman 1865, p 112) Rev Davis recorded that Maori believed that the European god had been responsible for the deaths. (Coleman 1865, p 112) Some

believed the European God had killed the believers and others believed the European God had killed the non-believers. (Coleman 1865, p 112) Davis himself believed that most Maori believed it was a judgement from God for the Europeans who were killed by Maori years ago.

Polio

Polio was initially known as Landry's disease and was first diagnosed around 1784. (Cumpston 1989, p 328) Over the years a number of polio types have been identified and the descriptions and naming of the various diseases associated with the polio viruses have undergone a number of changes. Today polio tends to be viewed as paralytic, as this strain of the disease is the most damaging. However, milder strains continue to exist and are often redefined as different diseases, such as aseptic meningitis. While it has generally been thought that polio was not present in New Zealand in the pre Treaty period, an epidemic in December 1835 that had been amongst Maori for eighteen months, (Coleman 1865, p 198) does suggest a form of polio. The disease began with a violent headache and moved to the glands before settling in the throat. Subsequently a violent fever developed. The throat closed and the patient could only swallow liquids with great difficulty. The disease passed from the throat to the shoulder and shoulder blades and most of the people affected died. (Coleman 1865, p 198) Davis described one family where the chief wanted medicines for his wife and children. Davis claimed the wife's mouth was closed with tetanus. As the rest of the family was also ill it would be unlikely that the woman was affected by tetanus, as tetanus is not contagious. The description does suggest that there was some type of paralysis. The closest matching description, based on the symptoms described, is bulbar poliomyelitis, which affects the brain, paralyses the nerves in the neck and throat and spreads down through the shoulders and back. The death rate from bulbar poliomyelitis is very high in comparison to other forms of poliomyelitis. (Swartout 1957, p 751 - 756) It was most likely that the chief's wife's mouth was closed by paralysis from the polio virus.

Measles

In August 1835 an outbreak of measles swept through the Maori population of the lower South Island. (McNab 1909, p 407) Tipu, son of Tahupaahi and Pikirauho, sister of Te Maiharanui, apparently contracted measles while in Sydney and brought

the disease back with him on the *Sydney Packet* in July 1835. The first affected were the Maori people at Measley Beach, so named because of the disease, in Otago. (Starke 1986, p xli) (Richards 1995, p 62) An expedition to Cook Strait had been mounted under Te Whakataupuka; however, the expedition was overcome by measles at Tokomairiro. Te Whakataupuka died, (McNab 1909, p 416 - 7) and Tuhawaiki his nephew became the prominent chief in the southern South Island. (McNab 1909, p 407 - 8) The exact death rate is unknown, however, in 1844 Tuhawaiki explained to George Clark that Otakou had been one the largest Maori settlements until measles had arrived one winter and whole families had sickened and died from it. (Begg 1973, p 178 - 9) (Richards 1995, p 63 - 4) The whalers also claimed that formerly Maori were quite numerous but there was significant mortality during a measles epidemic. (Shortland 1974, p 36 - 39) The whaling industry, which had been dependant on Maori labour, was seriously affected. (Begg 1973, p 179) As a result of depopulation, access to land along the southern coastline led to several small whaling stations springing up (Richards 1995, p 64) and the whaling station at Otakau being abandoned by 1840. (Shortland 1974, p 11) This altered the balance of power, land ownership and settlement in the region as the South Island had an early history of resistance by local Maori to the establishment of sealing and whaling stations along the southern most coast-line.

These epidemics of childhood diseases had varying effects in the population. There is no indication that mumps caused any loss of life, although the epidemic of whooping cough did. Bulbar polio, if that is what this disease was, causes a high incidence of death and this disease also appears to do so. The epidemic of measles in the South Island, however, appears to have caused considerable death and devastation.

As discussed in chapter two there were a number of old people observed by the early eyewitnesses, however, according to later evidence in certain regions this changed as a result of introduced infectious diseases. Some claimed that Maori generally recovered well from most introduced diseases, (Polack 1976, p 98) (Earle 1909, p 110) and Polack claimed that epidemic diseases did not have a significant impact on Maori. (Polack 1976, p 97) Dieffenbach claimed that in the interior where contact with Europeans was far less common the incidence of disease was much lower. (Dieffenbach 1843, p 22) However, others, such as Hobbs claimed that they did not

recover from European illnesses and died prematurely. (Williment 1985, p 118) Yate recorded that there were very few old people who would exceed the age of 50 by the 1830s, as war and diseases had taken their toll on the people, most especially the aged. (Yate 1835, p 163) It is therefore difficult to assess, without any accurate statistics, what impact these diseases had on the population and on the overall age of the population. However, these statements do imply that numerous deaths had occurred. It is usual for infections, especially influenza to hit older people especially hard, as the ability to combat disease wanes with age. It is therefore possible that infectious diseases did have a long term effect on the overall age of the population, especially in coastal regions where the populations were more exposed to the epidemics.

While it is difficult to assess the impact on morbidity and mortality of infectious diseases introduced into New Zealand by traders, whalers and other visitors, it would appear that a number of epidemics did arrive in New Zealand, most especially at the Bay of Islands. It is difficult to assess the impact of infectious diseases prior to 1808 due to the lack of evidence recorded at that time, however, epidemics across the Tasman would indicate that epidemics in New Zealand during the same period would have been typhus, typhoid and dysentery. An epidemic, probably of typhus in 1808, apparently killed many in Wangaroa. Another epidemic possibly occurred in the Hauraki region around 1815 but the evidence is inconclusive. A typhoid outbreak in 1832 caused a number of deaths, however, it is again difficult to assess the impact. The epidemic of catarrh in 1820 was carried down to Hauraki from the Bay of Islands region and spread as far south as Taranaki, apparently killing thousands in its wake. However, the 1827 epidemic of influenza did not have the same impact and most Maori apparently recovered quickly from it. this was not the case with the influenza epidemics that occurred between 1836 - 1838. There were apparently high death rates especially amongst the old people and those already in poor health. Mumps apparently had little impact although the whooping cough epidemic took many lives. A number of deaths in 1835 appear to have been as a result of polio, but the measles epidemic in the South Island in 1835 was devastating to the population. It is not known why the disease did not also occur at the Bay of Islands. It is therefore difficult to assess the exact impact on the population, however, it does appear that the epidemics occurred with greater frequency in areas with greater

contact with Europeans, which is to be expected. Presumably, these diseases did have some impact although the overall impact appears to have been transient as the diseases died out after completing their cycle. However, this was not the case for two other diseases, venereal disease, most especially syphilis, and tuberculosis. These two diseases, were to have a far greater impact on morbidity and mortality, and on Maori beliefs and practices.

What is worthy of note is that Maori believed the epidemics of typhus, catarrh, influenza and whooping cough were brought among them by Europeans and their atua. This suggests that they had not seen these diseases before, otherwise they would surely have had a theory of cause from an atua of their own. No mention is made in the literature as to whether Maori also believed that the epidemics of mumps and polio, which also occurred in the North Island were due to European atua. The measles epidemic in the South Island was believed by Maori there to have been brought in on the ship. Whether they believed it was a European atua or not is not clear. However, the idea that these diseases were of European origin dates back in the literature to 1808.

CHAPTER 4: VENEREAL DISEASE AND TUBERCULOSIS

The introduced diseases that appear to have had the greatest impact on Maori health and Maori society were venereal disease and tuberculosis. Tuberculosis and venereal diseases, most especially syphilis, share a number of symptoms such as eye infections, genito-urinary infections and joint pains. Because there is a significant crossover it is often difficult to differentiate between the diseases. Furthermore, the diseases possibly would have co-existed in some individuals. However, in some cases where a disease existed on its own or predominated identification can be more specific. Therefore, where the diseases are more clearly identified they are discussed separately. Because of the difficulty in differentiation reasonably detailed descriptions of the diseases have been provided in some instances.

VENEREAL DISEASE

Venereal disease was introduced into Northland and Queen Charlotte Sound from the first visits of the French and English explorers in the 1770s. The English had visited Queen Charlotte Sound several times during the 1770s; however, the new wave of Europeans to New Zealand did not appear to visit the area until the 1820s'. At Murihiku there was no evidence of any sexual liaison or introduction of venereal disease during the first voyages in 1770 or from the infrequent voyages in the early 1790s. The French visited Northland twice in the 1770s and recorded venereal diseases on the second voyage. However, mention of venereal disease doesn't appear in the literature again until 1814. It is therefore difficult to establish whether venereal disease had continued to affect Maori prior to 1790 or whether the diseases were reintroduced. Vessels began visiting Northland, the Bay of Islands and Murihiku from 1790 onward. There are very few journals for the period 1790 – 1814 and no mention is made of whether there were any sexual liaisons between the crew and Maori. Nevertheless, from the evidence it appears that venereal disease became more common in coastal villages than further inland where there had been little or no European contact or shipping. (Dieffenbach 1843, p 21) This would suggest that the diseases either re-established in the early 19th century or were contained in some way, as they did not become wide spread throughout the country.

Almost all of the evidence for venereal diseases comes from the Bay of Islands. As the Bay of Islands became a South Pacific resort for whalers and sealers it stands to reason that the incidence of venereal diseases was predominantly recorded there. The Bay of Islands does not typify the rest of New Zealand. The incidence of venereal disease there does not reflect the incidence of venereal disease anywhere else in the country. While there is evidence of sexual activity in other ports around the country, there is very little evidence in the literature to suggest a prevalence of venereal disease outside of the Bay of Islands. The prevalence of venereal disease would have also been exacerbated in the Bay of Islands by warfare in the 1820s and the huge increase in slaves brought into the region. Many young girls and young women, taken as slaves, were placed in sexual slavery. Whalers, sealers and traders had frequented the region for a number of years, although it was not until 1820 that descriptions of the industry began to come to light. Some of the women appeared to have formed relationships with the men and Captain Cruise recorded that several women lived on board the ship while it was in the harbour. (Cruise 1957, p 61) In other cases women went to sea with the vessels, but all ships had women on board in great numbers. (Watkins 1908, p 19) According to Markham in 1833 there could be four hundred to five hundred sailors in Kororareka looking for women at any given time. Most of the men were off whalers after being at sea for at least a year and many women spent three weeks or more on board the vessels while they were in the harbour. (Markham 1963, p 65) This continued and prolonged contact would have paved the way for a relatively high incidence of venereal disease.

The only way to assess the incidence of venereal disease is from eyewitness accounts and medical accounts of doctors who treated Maori or who treated crew for the disease. Diagnosis would have been difficult even for medical practitioners at the time as there was a lack of facilities, such as pathology reports and there was no recognition of micro-organisms at that time, therefore, diagnosis would have been strictly clinical. Early reports of venereal disease in 1821 from Dr Fairfowl and Richard Cruise on the *Dromedary* reported a reasonably low incidence of disease. (Cruise 1957, p 2187 - 8) (McNab 1908, p 555) (McNab 1908, p541) However, Ensign McRae, also from the *Dromedary* and Victor Charles Lottin in 1824 commented that venereal disease was prevalent. (Ollivier 1986, p 119) In 1827 M Quoy, a Russian Zoologist commented that 'several men reaped terrible consequences

from the women at the Bay of Islands'. (Wright 1950, p 226) The next reports on the incidence of venereal disease did not come until the 1830s. While Dr Fairfowl had recorded only 14 – 15 cases in 1821, presumably cases he had treated, by the 1830s reported incidence of the diseases had increased. For instance, Dr Watkins arrived in 1834 and commented that fifty women at Kororareka were affected by it, in total one fifth of the female population. (Watkins 1908, p 19 - 21) During his two month stay Dr Tawell had five or six cases under his care. (Tawell 1908, p 121) While the incidence of venereal disease may have been low in the early 1820s it had clearly escalated by the 1830s. This most probably occurred as a result of the increasing number of vessels arriving at the ports.

Most of the reports did not differentiate between syphilis and gonorrhoea, although most cases were probably syphilis. With gonorrhoea the incidence of disease would have been difficult to diagnose clinically as it can be asymptomatic in women, nonetheless there are a few reported cases that would indicate the disease was present. In 1827 the French vessel *Coquille* anchored at the Bay of Islands. Rene Primevere Lesson, the Pharmacist and Medical Assessor reported two cases of gonorrhoea among the crew on the journey to the Bay of Islands. (Ollivier 1986, p 159) Upon their departure there was one case of chancre, possibly as a result of syphilis, eight cases of gonorrhoea, (Ollivier 1986, p 161) and several cases of gonorrhoea amongst the crew developed after leaving. (Ollivier 1986, p 161) As the French were only in port for four days it is difficult to establish whether these cases of gonorrhoea were contracted at the Bay of Islands or whether they were contracted at Port Jackson. However, given the close proximity of the two ports and the frequent traffic, gonorrhoea was probably well established in both and traversing back and forth. According to Watkins gonorrhoea might have run its course, as it is a slight disease. (Watkins 1908, p 31) However, the impact of syphilis appears to have been far more devastating, especially in the early years of introduction.

The disease begins with genital sores leading to a general rash followed by ulceration and abscesses eating into bones and destroying the nose, lips and genitals, often ending in death. (Porter 1997, p 166) It has a primary incubation period of 9 - 90 days. According to Porter syphilis appears to have developed in the Americas around the time Columbus invaded. (Porter 1997, p 166) Marra, a crewmember of American

descent on the *Resolution*, discussed the effect venereal disease had had during the Spanish discovery of America. He described how hair and nails fell out and flesh rotted away from the bones. According to Diamond, syphilis arrived in Europe around 1495 and epidemiologically it behaved like a new disease when it was introduced. It was so virulent that it caused pustules from the head to the knees, resulting in the flesh falling from peoples' faces and leading to their death within months. By 1546 the micro-organism had adapted to what it is today. (Diamond 1998, p 210)

Houghton suggests there is a lack of evidence for syphilis in New Zealand prehistory and history. However, while the evidence does suggest that syphilis was introduced there is not a lack of evidence for its existence. Houghton quotes Dr Newman from 1881 on syphilis as evidence of low incidence, (Houghton 1980, p 135) however, Newman is not talking about incidence he is talking about virulence. Newman is comparing the severity of syphilis in the working class English with the severity of syphilis in Maori. By the time Newman is reviewing syphilis in 1881 the disease has been in the country for up to one hundred years. If the disease had reduced in virulence fifty years after its introduction into Europe, there is the possibility of reduced virulence and increased resistance in the Maori population. Therefore, this is not an argument for a low incidence of syphilis. Given the behaviour of the disease when it first emerged in America and when it spread to Europe, the possibility that the disease was also devastating upon its introduction to New Zealand is quite high. Reports from visitors to New Zealand would suggest that the disease was initially quite virulent. For instance, Dr Fairfowl in 1821 commented that venereal disease, presumably syphilis, at the Bay of Islands was very virulent and always resulted in death. (McNab 1908, p 555) Dr Watkins also described syphilis as a most destructive disease and very injurious to the constitution. (Watkins 1908, p 21) (Watkins 1908, p 31) Watkins claimed that at Kororareka especially, the women were affected by venereal disease of the most virulent kind. (Watkins 1908, p 19) There are a number of specific syphilis cases described in the literature, the earliest being from Nicholas in 1814. At Paroa he saw a woman whose face, hands and whole body were a mass of running sores and fetid ulcers. (Nicholas 1817, p 38 - 39) At Rangihoua he saw another woman who seemed to be happy, cheerful and vigorous, although her hands, face and body were in a state of livid ulceration. (Nicholas 1817, p 130 - 131) In

1820 McRae saw one woman who died and one who was dying of venereal disease. (McNab 1908, p541) Given that Watkins estimated the female population at Kororareka at approximately 250 and these visitors are only passing through, the incidence of syphilis could have been quite high.

An account recorded in 1827 at Murihiku suggests that syphilis had become established there and the impact of the disease was devastating. Boulton commented that it was a common disease among the people. (Starke 1986, p 63) According to Boulton the symptoms began with a 'wryness' of the mouth and a discharge from the eyes. The disease ravaged the face, eyes, nose, mouth, and lastly the hands and feet. The victim died in a half putrid state. One example was that of Te Wera, who was in his 30s and about 6 foot tall. He had travelled with Kent on the *Mermaid* in July 1823, presumably to Sydney. Three years later Shepherd described Te Wera as having a downcast mouth and Shepherd thought he was the ugliest man he had ever seen. (Starke 1986, p 63) Boulton described his face as 'scarcely human'. (Starke 1986, p 63) The case of Te Wera in the South Island is the only case found in the literature where the victim was a male.

While the disease is not gender specific women at the Bay of Islands appeared more likely than men to have been affected by venereal disease, as a result of sex slave trade. (Watkins 1908, 30) During the wars of the 1820s several hundred slaves were brought to the Bay of Islands. Many of the girls and young women were put to use in the sex trade and men often sold their services to the ships. (Watkins 1908, p 19) According to some observers most of the prostitutes were slaves who were degraded by the practice. (Marshall 1834, p 144) (Flatt 1908, p 44) These sex slaves were deliberately used to restrict the spread of venereal disease. Kendall commented that many young women contracted diseases from the seamen, (Elder 1934, p 82) and it is apparent that Maori were aware that venereal disease came off the ships. Dr Fairfowl believed Maori dreaded venereal disease calling it the Europe god (McNab 1908, p 555) and attributed venereal disease to intercourse with Europeans. (Watkins 1908, p 32) He commented that the spread of infection to the local community was checked because the infected persons were tabooed, (McNab 1908, p 555) and in 1824 Lesson recorded that Maori men did not allow their women to have relations with the men off the ships. Instead, they sent large numbers of slave girls onto the ships. (Ollivier

1986, p 161) By making these women tapu because they may have venereal diseases caused by the European atua, the disease was effectively contained. This was still an accepted practice in 1839 as Bright recorded that the natives at Kororareka were slow to take a wife because of the high level of prostitution. (Bright 1841, p 99) Maori therefore, actively sought to contain the disease within the female slave population as according to Lesson,¹⁸ Maori men considered any relationship with these women vile on religious grounds. (Ollivier 1986, p 161)

There may have been a number of reasons contributing to placing these women under tapu, and reserving them for the sex trade. First, Maori could supply the vessels with women, keep the sailors happy and continue trading. Second, with the needs of the sailors attended to, the wives and daughters of the chiefs and residents would have less concerns of molestation. Third by making the women tapu the disease was effectively contained. Whatever the reasons this practice would have restricted the spread of venereal disease at the Bay of Islands.

However, there was also another dynamic at play; evidence from both Murihiku and the Bay of Islands suggest that Maori men considered it an honour if their wives were popular and often took the women as wives after they ceased working on the ships. The men considered that the more sexual exploits the women had had the more attractive they must be. (Nicholas 1817, p 90) (McNab 1909, p 324) (Polack 1976, p 145 - 146) (Markham 1963, p 65) One example was Tara's wife, aptly nick-named Mrs Goashore, who had previously frequented the ships, although it is not clear whether she was a slave. (Nicholas 1817) According to Edwardson at South Cape a chief would be flattered if his wife received attentions from a white man, (McNab 1909, p 324) although a woman at the Bay of Islands was reserved only for her husband after marriage. (Polack 1976, p 145 - 146)

The practice of supplying slave girls to the ships would have contained the disease within a relatively small community, although marriages between chiefs and retired slave girls would have brought the disease into the wider community to some degree. However, in the Bay of Islands, if not at Murihiku, the sexual favours of wives were

¹⁸ Lesson was one of the first European visitors to specifically study and record his observations of Maori.

restricted to their husbands. Whether the husbands maintained their sexual relations within the marriage is not as clear-cut. Some chiefs had more than one wife. There was the possibility that there was some exposure to the general population, but in general the diseases were contained within a relatively small community.

Maori also practised a number of other methods that would have reduced the impact of the disease. Abortion or infanticide, tended to lessen the impact of venereal diseases. Whether this practice was employed for that specific purpose is unclear, however, the end result would have been a significant reduction in the number of children born with congenital syphilis. The practice was employed by the women who frequented the ships, as pregnancies as a result of relationships with European men appear to have been deliberately terminated. (Cruise 1957, p 187 - 8) (McNab 1908, p 539) The pregnancies were often terminated quite late (Cruise 1957, p 187 - 8) and this may have been due to the fact that pregnancy is an excellent contraception. It would not seem sensible to terminate a pregnancy in the early stages only to conceive again within a few weeks. According to Dieffenbach abortion was brought about by compressing the head of the infant before the birth was complete. Alternatively, a belt was wrapped around the stomach and violently compressed. Dieffenbach comments that many stillbirths were the result of this practice.¹⁹ (Dieffenbach 1843, p 26) Whatever the reasons, the result of the terminations would have been a reduction in congenital syphilis, whether that was the intention or not.

When the women become aware they had venereal disease they left the ships and returned when they were cured. (McNab 1908, p541) (Bright 1841, p 99) The impact of the diseases was reduced as a result of this method. (Polack 1908, p 89) (Bright 1841, p 99) Thus Bright commented that syphilis resulted in few deformities. (Bright 1841, p 99) The cure involved the use of a steaming method to treat venereal disease with indigenous plants that were known to the natives. (Polack 1908, p 92) (Polack 1976, p 98) (Bright 1841, p 99) The method was to lay the herbs over hot stones and pour hot water over them, however, according to Dieffenbach the 'inferior classes' made steam baths by infusing herbs in boiling water. (Dieffenbach 1843, p 61) The water was heated by dropping hot stones into an ipu, or trough. If treatment was

begun when the primary lesion first appeared and the treatment was effective in helping to clear the lesions, it would have helped reduce the spread of the disease as it is most often spread through contact with the primary lesions, which are most often on the genitals.

In conclusion, while venereal disease, especially syphilis, would have had a long term impact on Maori, attempts by Maori at the Bay of Islands would have been reasonably successful at containing the disease, and the virulence of the disease would have reduced over time as it did in Europe.

TUBERCULOSIS

Tuberculosis was having an impact on Maori health as early as 1808. While contemporary scientists are able to identify and classify the micro-organisms responsible for various forms of tuberculosis today, there was no clear identification, or even knowledge of micro-organisms in the period prior to 1840. Descriptions of the disease were often obscure references to symptoms, or were identified as consumption, atrophy, scrofula, phthisis, burst blood vessels, spitting blood, violent cold or pneumonia ending in consumption. Observations such as Dr Tawell's that numerous cases of pulmonary tuberculosis developed out of scrofula (Tawell 1908, p 121) reflect the lack of knowledge of the disease at the time. To understand the process and diversity of the disease, as it existed in the early period, a description of the forms and symptoms that the disease can manifest is included. The following description of tuberculosis is taken from a medical guide written in 1957, (Swartout 1957, pp 796 - 810) and a pathology textbook written in 1995. (Chandrosoma P & Taylor C. R. 1995) The descriptions, despite an intervening period of nearly forty years, differ very little. ²⁰

Tuberculosis is not always an acute disease; it can be chronic or latent. The causative micro-organisms can live outside of the body for several days under certain conditions; therefore contact with the disease can be quite widespread. Spitting is one

¹⁹ It is difficult to differentiate between abortion and infanticide in these cases. They were either very late abortions or early infanticide, as it appears the child was not fully born before its life was terminated.

²⁰ The incidence of tuberculosis in first world countries has considerably diminished as a result of public awareness, improved hygiene and sanitation, and the use of antibiotics, however, tuberculosis has never been fully eradicated and new antibiotic resistant strains are emerging.

way in which the micro-organisms can be rapidly spread, however, they can also be spread by dust, sneezing, coughing, hand to mouth passage and drinking vessels. The disease can also be spread from unpasteurised cows' milk and direct person-to-person contact. In the early stages an infected person may not be aware that they have the disease and it can be passed from person to person, perhaps within a family, before anyone becomes aware of its existence. Tubercles containing the micro-organisms may form within the lung and be destroyed by the immune system before the disease ever makes its existence known. The tubercles can also remain latent in the lung for long periods before making their presence known. In addition, tuberculosis can have periods of inactivity and then resurface again. Individuals are most vulnerable to the disease in their teens and early twenties or if their immune system is also dealing with other infections such as influenza. The disease has many manifestations but two common factors that characterise the disease are exhaustion and significant weight loss, these symptoms led to the labels consumption and phthisis. Other warning symptoms are coughing, spitting blood, sharp pains in the chest, fever and night sweats. Older persons may appear to have bronchitis, asthma or even heart disease when in fact they have chronic tuberculosis, as all of these symptoms generally indicate pulmonary tuberculosis.

As a result of the epidemics of influenza, catarrh and other infectious, especially respiratory, diseases that swept through Maori communities with increasing frequency in the pre treaty period, tuberculosis was able to gain a strong hold on its victims. These circumstances coupled with fatigue, cold and hunger and other forms of stress only served to exacerbate the disease.

Incidence of Pulmonary Tuberculosis

The earliest recorded case of pulmonary tuberculosis in New Zealand, that of Matara, dates back to 1808. However, cases may have occurred much earlier as there is evidence that there were a number of cases of tuberculosis on the first voyages of the French and English to New Zealand. (Hanham 2001) Whether Maori were infected with tuberculosis from these early voyages is difficult to ascertain, as there is an absence of literature for the period 1780 - 1805. After 1820 the incidence of tuberculosis appears more frequently in the literature with Cruise, (Cruise 1957, p 193) McRae, (McNab 1908, p 541) Dr Fairfowl (McNab 1908, p 555) and Rene

Lesson (Ollivier 1986, p 160) all commenting on the high incidence of the disease. This increase can in part be attributed to an increase in European observers and a subsequent increase in literature, however, there also appears to be an increase in actual incidence of disease. There are a number of factors that contribute to the increase including the wars and displacement of the 1820s, the consequent periods of famine, the intensive over production of prepared flax to purchase muskets, the shift from hillside pa to huts built in damp swampy areas, the shift to a diet more reliant on European foods, especially the potato, and the greater exposure to the outside world that brought increased exposure to tuberculosis and introduced periodic epidemics of other, especially respiratory diseases. All of these combined contributing factors created greater challenges to the previously healthy immune systems of Maori, which succumbed to the increased demand. Tuberculosis, a latent and opportunistic disease, was able to gain a foothold on Maori society that would not be brought under control until well into the twentieth century.

From 1827 reports of the highly fatal impact of tuberculosis began to appear, (Earle 1909, p 110) and by 1829 William Williams had recorded that tuberculosis was killing many of the natives. (Williams 1867, p 118) Reports of the disease and the associated death rate continued to escalate through the 1830s. (Coleman 1865, p 168) (Yate 1835, p 283- 304) Claims that Maori were a dying race began to emerge in the late 1830s and were directly attributable to the high incidence of deaths from tuberculosis. (Markham 1963, p 66) (Williment 1985, p 118) (Buller 1878, p 170) (Polack 1976, p 61) Although there is limited evidence from the South Island Shortland commented that tuberculosis had killed many and was a very well established disease. (Shortland 1974, p 65)

Laryngeal Tuberculosis

Laryngeal tuberculosis is characterised by hoarseness, cough and painful swallowing. There is only one recorded case of probable laryngeal tuberculosis and that was Matara, son of Te Pahi. He travelled to England on the *Buffalo* with Governor King in 1807 and in 1808 arrived back in Sydney. He developed hoarseness in his throat that settled on his lungs and died a few months later. (McNab 1914, p 120)

Scrofula

While pulmonary tuberculosis is widely recognised, tuberculosis can affect many other areas of the body. If the tuberculosis bacilli overwhelm the immune system the micro-organisms can spread to the lymph glands. Lymphatic tuberculosis is usually seen as swelling of the glands in the neck, which have ulcerated and ooze pus. Other glands throughout the lymphatic system can also become infected. This was not widely recognised in the early 1800s, consequently, scrofula, ulcerations, abscesses, cutaneous diseases and lymphatic swellings all appear to describe scrofula in the literature. The descriptions varied from author to author and were not always dependant on the medical knowledge of the observer. There does not appear to be a clear diagnostic differentiation for the period. With the lack of knowledge of micro-organisms at the time, the lack of understanding of immunity and the lack of pathological analysis the causative agents are not easily identified. However, as there appears to have been a very high incidence of tuberculosis in the pre-Treaty period it is more likely that these skin afflictions are various stages of scrofula. Scrofula was also mistakenly described as leprosy or venereal disease. While venereal disease and leprosy could produce skin lesions there are a number of indicators that help to differentiate the diseases. It has been established that leprosy was not introduced into New Zealand until after 1840, (Montgomerie 1988)²¹ therefore any descriptions of leprosy prior to 1840 are probably scrofula. Venereal disease and scrofula can be difficult to differentiate, however, scrofula or lymphatic tuberculosis generally begins with ulcerations over glands, especially in the neck, venereal disease or more especially syphilis, begins its primary stage with a lesion on the genitals, however, in the second stage lesions erupt on the torso, face and hands. Advanced scrofula and advanced syphilis could produce similar lesions. For this reasons descriptions that are difficult to differentiate are described in combination and there is every possibility that the two diseases could occur simultaneously in any given individual. However, the evidence for scrofula will be discussed first.

As early as 1815 Kendall described scrofula among Maori, commenting that Maori were prone to the disease. (Elder 1934, p 82) (Elder 1934, p 86) By the 1830s

scrofula was prevalent, the ulcerations were observed to come and go and were described as 'malignant'. (Watkins 1908, p 20) (Marshall 1834, p 144, 261) (Watkins 1908, p 13) (Watkins 1908, p 31) (Polack 1976, p 98) (Wilkinson 1908, p 102) According to Tawell hundreds were affected with the disease. (Tawell 1908, p 119) (Tawell 1908, p 121) Once again there is limited evidence for the South Island, nonetheless according to Edwardson in 1824 many at Murihiku suffered from scrofula, (McNab 1909, p 325) and from the 1830s swellings in the neck, hips and sides were a major form of disease. (McKay 1873, p 25)

Aside from scrofula there is another form of tuberculosis that manifests on the skin, which is called dermal tuberculosis. It attacks the nose and face especially, but can appear anywhere on the skin. Small soft tubercles appear on the skin that may spread and ulcerate before healing, leaving disfiguring scars. This disease is sometimes known as lupus vulgaris. Polack claims he saw only one case of leprosy, (Polack 1838, p 275) however, it may have been dermal tuberculosis, which is relatively rare.

Dropsy

The swallowing of sputum can cause micro-organisms to spread to the intestinal tract resulting in intestinal tuberculosis. The infection is characterised by diarrhoea, or alternate diarrhoea and constipation, abdominal pains and tenderness. There is often blood in the stools. The intestines can become so inflamed and damaged that partial or complete blockage of the bowel occurs, and most often ends in death. Intestinal tuberculosis was often called dropsy in the 1800s, because the stomach swelled. According to Polack dropsy was very rare among Maori, (Polack 1976, p 98) however, Polack himself recorded that inflammatory diseases of the stomach were frequent. (Polack 1976, p 97) (Polack 1976, p 98) There does appear to be a number of descriptions of disease that could have been intestinal tuberculosis. For instance, Dr Fairfowl saw inflammation of the bowels, dysentery and colic, (McNab 1908, p 555) and Dieffenbach described irritations of the 'mucous membranes of the intestinal canal and other mucous membranes' (Dieffenbach 1843, p 21) and gastric fevers. (Dieffenbach 1843, p 21)

²¹ Montgomerie has written an excellent analysis of why leprosy was not a disease of the early 1800s. The reader

Rheumatism or Tuberculosis

In the 1800's rheumatism was a term generally applied to a range of aches and pains and did not specify any particular disease. The bones and joints, especially the spine can be affected by the tuberculosis bacilli. A lack of treatment can cause bone deformities and can also lead to general infection and death. Therefore, cases of rheumatism described in the literature were possibly not rheumatic fever, but tuberculosis. Dr Fairfowl, 1821, commented that he saw rheumatism amongst Maori but does not specify what form this took. (McNab 1908, p 555) Polack saw one man with a spinal protuberance. (Polack 1838, p 273) Both of these descriptions were probably cases of tuberculosis. Tuberculosis of the joints would have been especially so in cases where there were other symptoms indicating tuberculosis, such as sore eyes. In 1820 Cruise commented that 'Consumption, violent Rheumatism, and sore eyes, seem to be the prevailing disease; many die of inflammation of the lungs and bowels'. (Cruise 1957, p 193)

Meningeal Tuberculosis

Tuberculosis can also spread to the central nervous system and once in the central nervous system it can cause a form of meningitis that is slow, progressive and usually fatal. The majority of cases occur in childhood or early teens. The patient becomes apathetic and drowsy, with disturbed sleep and a headache that causes intermittent screaming. Delirium and a range of other symptoms that differ from patient to patient appear late in the course of the disease. The forehead becomes contracted, the facial expression is blank, the pupils are contracted, the eyeballs blood shot and thickly covered with mucous, and the abdomen is tight and retracted. The patient lies on their side in a foetal position, paralysis appears and the patient sinks into a coma and dies. Meningeal tuberculosis is usually secondary. However, Cruise described the cases of Perahiko and Cowerapopo [sic]. Perahiko was an elderly man and suffered some 'rheumatic' complaint that had caused the loss of the use of his limbs. (Cruise 1957, p 32) According to Cruise Perahiko was racked by agonising pain upon his death. (Cruise 1957, p 198) Perahiko died before Cruise left New Zealand. (Cruise 1957, p 85) Around the same time at Waikino, a chief by the name of Cowerapopo [sic] was an elderly man who had lost the use of his limbs from a 'rheumatic' disorder. (Cruise

is referred to his analysis for further information.

1957, p 98) Meningeal tuberculosis was probably occurring as early as 1815 as Kendall claimed that Maori were subject to cutaneous diseases and were often afflicted with violent headaches and bad eyes. (Elder 1934, p 82) At least one had a 'violent cold'. (Elder 1934, p 86) These are all symptoms of tuberculosis. While certain forms of tuberculosis may affect children more often than adults, the principle that has been applied to childhood illnesses can also be applied to tuberculosis. When the disease is new to the people, that is when the population is immunologically naïve the behaviour of the disease will be different to the behaviour of the disease in a previously exposed population. Therefore, tuberculosis was probably a relatively recent introduced disease and may have been in the country for only a few years when Kendall and Cruise were recording their observations. However, the evidence from 1815 suggests that there were a number of deaths from tuberculosis in the period around 1815 and many of these cases appear to have succumbed rapidly to the disease. This would suggest that exposure to the bacteria was relatively recent and the population was immunologically naïve. Disseminated tuberculosis is where the disease spreads through the body causing high fever, rapid weight loss, exhaustion, profuse sweating, rapid pulse, headache, dizziness, dry tongue, distended stomach, anaemia and death usually within two to six weeks

Genito – Urinary Tuberculosis

Tuberculosis can also affect other areas of the body such as the kidneys and bladder. The symptoms are frequent and painful urination, and albumin and pus may cause cloudy urination. A common form of tuberculosis in males is epididymis or testicular tuberculosis, initially affecting only one gonad but spreading to the other. Symptoms include swelling, tenderness, painful ejaculation and post coital weakness. The disease usually also involves the kidneys, bladder and urinary tract and other areas of the body. In the female the fallopian tubes can be affected with symptoms of inflammation and infection. Fallopian tuberculosis is usually secondary to intestinal or peritoneum infection. The incidence of these forms is difficult to assess, as symptoms may have been mistaken for venereal diseases, however, this form may have been relatively common as a number of remedies were employed to treat genito-urinary infections. According to Polack, kawakawa was useful for complicated urinary complaints and was used in the vapour bath method, described above. (Polack

1976, p 99) Polack also records the use of kaikatoa, or manuka for the use in the more 'disgusting of diseases'.²² (Polack 1838, p 277) (Polack 1838, p 395)

Venereal Disease and/or Tuberculosis

Many missionaries do not seem to mention scrofula. As discussed above there was some difficulty in differentiation between scrofula and syphilis. Missionaries also appeared to avoid the plight of the young women on the ships; it is therefore possible that the missionaries believed that syphilis and scrofula were the same thing. However, the evidence does suggest that many of the young women were suffering from both diseases. Marshall commented that many of the prostitutes have scrofula and 'a terrible disease of much less doubtful origin' the combination of the two diseases has 'woeful consequences'. (Marshall 1834, p 144) Marshall attended to 'several frightful cases'. (Marshall 1834, p 144) In one instance a woman's chest was bare of flesh as a result of ulceration that had exposed the breast-bone and some ribs. (Marshall 1834, p 144) Watkin also suggested that venereal disease in a person with scrofula would be more severe. (Watkins 1908, p 30) Dr Marshall who was at the Bay of Islands for a month in 1834 commented that this 'terrible disease' was often fatal at the coastal villages. Marshall claimed that many infants imbibed the virus from their mother's milk and were 'dreadful sufferers for their parents sin'. (Marshall 1834, p 144) In March 1835 Henry Williams recorded that they saw many cases of venereal disease everywhere they went and children were born with it. He commented that he saw one sad case, one of Te Wherowhero's little boys, with the disease. (Rogers 1961, p 428) While Williams claimed that he saw venereal disease it is difficult to know how he was able to diagnose the disorder. As described above the disease was most likely to have been scrofula.

There is evidence of venereal disease and tuberculosis in Murihiku from the 1820s; however, there are only a few accounts. Captain Edwardson commented that the principal diseases were elephantiasis and pian (yaws). (McNab 1909, p 325) It is more likely that elephantiasis is the swollen testes sometimes caused by either gonorrhoea or tuberculosis and pian is the ulcers caused by syphilis. Both the micro-organisms for elephantiasis and yaws are tropical and the South Cape is not a tropical

²² Polack records that kaikatoa was used in a decoction by both Maori and Europeans; it may therefore be of

climate. Edwardson goes on to comment that many have lost their hands and feet, their bodies were extremely thin and their extremities rot away. This would tend to indicate a combination of the diseases and a lack of resistance to both.

Dieffenbach commented that where exanthematic diseases such as rashes and skin eruptions such as smallpox existed, or where constitutions and resistance were challenged, venereal disease could develop serious complications such as diseases of the hip joints, the spinal column and spinal distortion in infancy and these complications were becoming increasingly common. (Dieffenbach 1843, p 21 - 22) However, the descriptions offered here by Dieffenbach may not be congenital venereal disease, presumably syphilis. Spinal distortion and involvement of the spinal column was more likely to have been tuberculosis. While congenital syphilis can cause lesions on the bone, children born with active syphilis are more likely to be stillborn or born prematurely with enlarged liver and spleen. (Waseem 2002) These children would not have been likely to survive in early Maori society. Congenital syphilis can develop in a child who was born asymptomatic, however, the literature does not describe spinal curvature, distortion, or hip dysplasia. Furthermore, as the women who frequented the ships were predominantly slaves they were also very undernourished. The literature also indicates that there was a high rate of abortion and infanticide amongst the women who frequented the ships. It is therefore questionable whether any infected infant would have survived. For a child to be born with congenital tuberculosis, a woman would have to have tuberculosis of the placenta or birth canal. (Batra 2002) Given the high rate of prostitution amongst young slave women this was a possibility, however, a newborn infant could easily contract tuberculosis from close contact with its mother. Given the high incidence of tuberculosis by the time Dieffenbach was in the country it is more likely that the children he described were suffering from tuberculosis.

The impact of these two diseases appears to have been more devastating than the impact of other infectious epidemic diseases, in part because they are chronic diseases. Despite the difficulty in identifying the diseases from early eyewitness evidence a number of forms of tuberculosis were identifiable in the literature by their

European origin. Kawakawaa and the vapour bath method, however, appear to be unique to Maori.

symptoms. While symptoms of venereal diseases were a little easier to extricate from the literature there was still some difficulty in differentiation and there were a number of cases where both diseases possibly coexisted. Both the diseases appear to have been introduced in the early 1800s. This would suggest that they did not become widely established after first contact with the early French explorers in the late 1700s. Attempts by Maori to control venereal disease appear to have had some degree of success; nonetheless the disease would have had a significant impact.

CHAPTER 5: MAORI VALUES AND BELIEFS REGARDING DISEASE

This chapter explores the development of ideas regarding disease causation from traditional beliefs about familial atua through to the development of the cult of Papahurihia to assess the impact introduced infectious diseases might have had on Maori values and beliefs regarding disease. Traditional Maori society had a well developed belief system that attributed misfortune and illness to the actions of atua. The first reaction of Maori to the introduction of infectious diseases was to expand their existing belief system to encapsulate an understanding of infectious disease causation. Some Maori believed that the increasing number of infectious diseases were due to indigenous atua becoming very angry that they had allowed white men in. (Craik 1830, p 231 - 232) They believed that Europeans were under the influence of their own, different atua as their illnesses were less severe. (Williment 1985, p 36) This theory followed along traditional ideas that familial atua punished their own for transgressions. However, this was not the only theory. The expansion of beliefs appears in the literature from as early as 1808, when Maori at the Bay of Islands claimed an epidemic of typhus was caused by European atua and a belief also developed that disease was transmitted by ships or people. This chapter explores the possibility that the atua known as ngarara or the lizard atua can be attributed to the arrival of tuberculosis. By 1820 some Maori were claiming that it was the European God and his priests the missionaries who were causing disease. Out of this some Maori developed the idea that the missionaries must therefore be sorcerers. Therefore, Maori attempts to understand and enculturate introduced disease will be explored in this chapter. A special emphasis will be placed on how Maori attempted to explain tuberculosis, a disease that made rapid inroads into Maori health and explores the possibility of a connection between the multiplicity of ideas and the emergence of the cult led by Papahurihia in the 1830s.

Traditional Beliefs

Maori attributed afflictions to the actions of atua. Familial atua were believed to be the wairua (spirits) of ancestors who had died. These ancestors were believed to return to the human world to oversee the actions of their relatives or to punish others who transgressed their tapu. They could provide both protection and punishment to their descendants. According to Dieffenbach atua were 'the secret powers of the universe, whether they appear to them [Maori] as beneficent or malignant.'

(Dieffenbach 1843, p 118) In the traditional literature disease,²³ probably genetic disease, was ascribed to atua as punishment for a breach of tapu, alternately it was thought to be the result of an act of makutu (sorcery), (Dieffenbach 1843, p 58) which also involves atua. Shortland commented that the cause of disease was believed to be atua punishing a transgression. (Shortland 1974, p 30 - 31) It was generally recognised that atua could take forms such as birds, (Polack 1976, p 241) lizards, clouds or a ray of sun. (Dieffenbach 1843, p 117) In these forms atua represented warnings of impending danger or indicators of good fortune. Their presence was generally made known by certain signs such as twitching of the limbs or dreams or unusual incidents. These signs were read and interpreted by the tohunga (priest). His or her job was to ascertain the meaning of the signs and effect a remedy. According to Polack preventative measures such as dedicating a lock of hair to an atua to dissipate or dispel diseases and afflictions were commonly employed. (Polack 1976, p 234) One class of familial atua was considered to be particularly malevolent as they had not had the time to develop a close bond with family members before departing this earth and therefore had no qualms about visiting punishments on family members. This class of atua were known as kahukahu. (Shortland 1856, p 115) However, to some sectors of Maori society the actions of indigenous atua in general appeared to become more malevolent. As epidemics began to occur debate arose within Maori communities as to whether indigenous atua were becoming angry because of the increase of Europeans in the settlements; or whether the atua were European and had arrived as a result of allowing Europeans to settle amongst them.

European Atua

Some Maori began to point the finger at European atua as the cause of increased disease. They claimed that before the whites arrived young people did not die, but lived to be so old they crawled on their hands and knees. (Craik 1830, p 231 - 232) Now they claimed that many were dying from diseases brought amongst them by Europeans. (Craik 1830, p 231 - 232) (Polack 1838, p 235) As discussed in chapter three the belief that the European atua were causing epidemics of disease appears in the literature from as early as 1808 - 1809, after a probable typhus epidemic at Whangaroa. This European atua came in the form of epidemics and diseases such as

²³ In Murihiku Edwardson associated Rakiora with diseases. Rakiora is not a familial atua but comes from another

cholera²⁴, influenza, measles and another more deprecating disease²⁵. (Polack 1838, p 234 - 235) (Polack 1976, p 246)

Along with the belief that European atua were the cause of disease there developed a theory of disease transmission, with a number of Maori claiming that Europeans or European vessels brought the diseases amongst them. This theory of disease transmission dates back to the arrival of Captain Cook where an epidemic is said to have devastated East Coast tribes after the arrival of a European vessel. (Dieffenbach 1843, p 13 - 14) Another is said to have swept through Queen Charlotte Sound around the same period. (Taylor 1974, p 207) These beliefs continued to appear throughout the period, for example, when Earle arrived at Hokianga in 1827 an influenza epidemic was raging through the population and Maori claimed they had caught it from Europeans. (Earle 1909, p 110) Similar accusations arose in the South Island in 1837 when Captain Bruce of the *Sydney Packet* reported that the natives were restricted in their raids on Te Rauparaha by a severe attack of influenza. Bruce claimed his whole crew was affected, and that the natives threatened to kill him for bringing it among them. (Carrick 1903, p 13)

As discussed in chapter four venereal diseases were also attributed to European gods, however the evidence suggests that religious practices were employed in an attempt to control the disease. With the advent of shipping and the arrival of venereal diseases, women who frequented the ships and were exposed to venereal diseases were made tapu. It was traditional in Maori society for a woman to be put under tapu as a result of her sexual status, either as a puhi (virgin) or as a married woman. Placing the women under tapu indicates that religious practices were expanded to encapsulate the advent of venereal diseases. There does not appear to have been any specific religious practices associated with introduced epidemic diseases, although as discussed in chapter three a number of practical remedies were employed to alleviate the symptoms. However, there seems no reason to think that the traditional practice of isolating sick people, and designating them as tapu, did not continue.

class of deities.

²⁴ The term Cholera appears to have been used to describe Typhus. There are no recorded epidemics of Cholera, although Typhus epidemics did occur frequently.

The European God

The belief that infectious epidemic diseases were caused by European atua in response to introduced disease reflects a traditional view of gods as multiple. This began to undergo a change in response to introduced disease. A belief began to emerge that it was the European God that was to blame, although the theory that disease could be transmitted by Europeans or vessels, as discussed above, lingered. This belief first appears in the literature in 1820 when an epidemic of catarrh at Hauraki that spread through to Taranaki was attributed to Marsden and the English God he brought with him. In addition, Maori at Waikari told Reverend Chapman not to visit them again as before his visits they had been in good health and since his visits began they had become sick and unwell. (Marshall 1834, p 268)

The belief that these diseases were caused by God probably arose as a result of missionary preaching. An epidemic of whooping cough occurred in 1828. Maori asked Henry Williams many questions about illness during the whooping cough epidemic. Williams told them it was God's anger and they could not expect anything else for their wickedness. He insisted they must convert to Christianity and hoped the illness would persuade them. (Rogers 1961, p 31 - 32) Davis recorded that some Maori believed that God had killed the believers, while others believed God had killed the non-believers. (Coleman 1865, p 112) Davis himself thought that most Maori believed the epidemic was a judgement from God because of the Europeans killed years ago by Maori. (Coleman 1865, p 113) While this shows that traditional ideas about revenge continued to operate, nevertheless, Davis' evidence shows that God has been incorporated into the Maori belief system, and suggests that Maori viewed this new God from a traditional perspective. Davis' view is evidence that both Europeans and Maori had religious views of causation, and suggests that it would be easy for Maori to make the step to blaming the European God for the increase in disease. Because Maori atua were activated through ritual, the European God was believed to act through the praying of the missionaries, and Hall, King and Kendall all claimed that Maori believed their continual praying was making the European God destroy them. (Elder 1934, p 111) Kendall had commented that Maori were ignorant of the causes of their diseases, as they believe it is always an atua

²⁵ Polack will be referring here to Venereal Disease.

giving them pain. Kendall believed that a sense of guilt and shame needed to be instilled in Maori. (Binney 1968, p 75) Butler set one chief straight explaining that the New Zealand atua was nonsense. The man's illness was brought about by his improper conduct. Butler explained that many diseases were brought about as a natural consequence of improper conduct. He explained that human bodies are subject to disease and death on account of our doing bad things against the commands of the great 'Atua'. (Barton 1927, p 218 - 219)

Nevertheless, as early as 1815 the impact of Christian teaching had created a dichotomy between God as the cause of disease (Elder 1934, p 111) and an alliance of indigenous atua with the biblical devil since the arrival of the missionaries. (Binney 1968, p 128n) (Elder 1934, p 111)

The Entrail Eating Atua

Pulmonary tuberculosis caused the greatest consternation amongst Maori, this was often expressed as the belief that atua were eating them from the inside. This atua appears in the observational literature as a ngarara or lizard and these terms are loosely used by the eyewitnesses. More correctly, ngarara is a generic class of land crawling creatures, including lizards. According to Earle, at the Bay of Islands the lizard was sacred and never killed or injured. Carvings on buildings depicted the lizard taking man by the top of his head, the tradition being that that was 'the origin of man'. (Earle 1909, p 117) Earle recorded that when the world was created a man in a canoe was fishing and pulled up a fish. The fish became the land and then a lizard crawled onto the land. The lizard then pulled man up onto the land by his hair. (Earle 1909, p 211) Best also records that lizards have come up onto the land from the sea, as they were the children of Tangaroa who sought refuge on the land. They are considered to be the children of Punga, and include all species of lizard, including tuatara. (Best 1924, p 115) In other traditional literature throughout the country, ngarara are apparently reptilian monsters, renowned for capturing women and taking them away to make wives of them. Usually, the ngarara is outwitted by the women or their families and the ngarara is defeated. (Tremewan 2002, p 219) (Orbell 1995, p 245) Best records that the lizards were used as guardians of sacred places such as burial caves, (Best 1924, p 132) but they can be evil omens if they are someone else's

atua. (Best 1924, p202) In none of these situations is it recorded that the atua enter human beings and eat their entrails.

Best records that Whiro is believed to be the cause of disease and he often appears in lizard form. Best also argues that Whiro was believed to consume the souls of men. (Best 1924, p 116) Best uses Cruise as his source for Whiro as the entrail eating atua, however, Cruise does not say this. Cruise said that at the point that the person was believed to be incurable the person was believed to be afflicted by an atua. The atua took possession of the person in the shape of a lizard and devoured the intestines. Cruise makes no mention of Whiro, however, this is not surprising given Cruise's relatively slight acquaintance with Maori. Best also relies on Yate's assessment of Whiro. Yate claims Whiro eats the souls of men and aligns Whiro with the devil. Yate is therefore trying to express Maori thought in European concepts. This is inaccurate. There is a huge difference between the soul and the intestines. The association between tuberculosis and entrail eating atua is literal not metaphorical. These are Best's only two sources for suggesting the entrail eating atua is an indigenous concept and does not constitute a strong enough argument. Tyerman and Bennet recorded an incident in 1829 of a tohunga with tuberculosis. The man told them his people believed his disease was due to God or rather the devil being within them and devouring their hearts. (Montgomery 1831, p 139) The man had been placed under tapu. It is particularly significant that this man identified God and the devil. As discussed above this was Yate's premise. It would appear that Whiro, the devil and lizards all form part of a confused understanding of the causes of disease that encapsulate both traditional and Christian ideas.

The evidence suggests that Maori believed that these atua were also of European origin. There are a number of grounds in support of this belief. The belief that atua in lizard form were the cause of death shifts away from the more traditional beliefs regarding lizards in the Bay of Islands, where traditionally the lizard had been associated with life not death.

In addition, the idea of tuberculosis in ngarara form is only found in the Bay of Islands at this period in time and this would indicate that it was not a universally

traditional concept.²⁶ Maori who died of tuberculosis early in the contact period, or at least those recorded in the literature, had all been outside of New Zealand and had for the most part spent time in the missions while either in Parramatta or England. This is particularly significant given that the first case of tuberculosis blamed on an entrail eating atua is that of Ruatara, a close ally of Marsden and the other missionaries who arrived in 1814. This case has special significance as the disease developed special attention from both Maori and European from this point.

Ruatara, was nephew to Te Pahi, whose son Matara died of tuberculosis in 1808. No mention was made in the literature that Matara had died as the result of an entrail eating atua. Similarly, Mowhi [sic] was also suffering from tuberculosis; however, his death was not attributed to an entrail eating atua. While still in England Ruatara believed his disease, which manifested as symptoms of coughing blood, was caused by the beatings he had received while working on a whaling ship. He therefore, did not believe at that point in time that his disease was a result of an entrail eating atua. This would suggest that the association between tuberculosis and entrail eating atua had not been established at that time.

Marsden was among the first to record Maori views on diseases and noted that afflictions were believed to be caused by 'superior beings'. (McNab 1908, p 385) Marsden was among the first to be blamed for bringing the European God amongst Maori at Hauraki in 1820. Marsden was also at Ruatara's beside along with Thomas Kendall and John Nicholas. This raises the question of whether Christian concepts had already been introduced to Maori, especially the ideas that God caused disease and that the serpent was evil. Ruatara spent several months with Marsden at the mission in Parramatta, as did other Maori from the Bay of Islands. It is probable that they were introduced to Christian concepts while there. Best was writing during a period when diseases such as tuberculosis were believed to be indigenous. This thesis

²⁶ The only indication that South Island Maori had any particular views on the causes of tuberculosis, is in the form of Taipo. ²⁶ This deity appears in the South Island literature in 1827. There is only one example, which is that of Kokoro, son of Te Whakataupuka, who had complained sometime earlier of pain in his breast and died on Codfish Island while still a young man. It was believed a 'Taipo' had killed him. Starke, J. (1986). Journal of a Rambler: The Journal of John Boulton. Auckland, Alexander Turnbull Library Endowment Trust, Oxford University Library.

Anderson, A. (1998). The Welcome of Strangers: An Ethnohistory of Southern Maori 1650 - 1850. Dunedin, University of Otago Press.

has established that it is not. It is therefore possible that the entrail eating atua developed as a result of a number of influences, both indigenous and Christian and was created as indigenous by early writers, such as Best.

According to Binney ngarara assume significance in Kendall's letters around 1824 in association with the biblical serpent. However, Kendall was discussing ngarara or lizards from as early as 1815. For instance, Te Waimuri was so ill and close to death he had been declared tapu. He believed a reptile god had entered his body and he would die. Kendall pointed out to him the 'ignorance' of his beliefs and explained to him that all men had offended the great Atua to which Te Waimuri pointed out that all men must therefore die. (Elder 1934, p 86) When Te Waimuri died 23 July 1815, Kendall commented that his lungs were affected. (Elder 1934, p 87) In addition, Whare, who was also ill in 1815 had been coughing and spitting for many months. He had been put under tapu and was afraid the atua would kill him. (Elder 1934, p 86 - 87) One man believed an atua was inside him and eating his vitals. (Elder 1934, p 86) Kendall told him there was only one Atua and all men had offended him so all men must die. (Elder 1934, p 87) This evidence would suggest that there may be some elements of traditional belief, however, these are cases of tuberculosis. A pre-contact belief that tuberculosis was caused by entrail eating atua could not have existed prior to contact. Furthermore, chapter two established that there was a very low incidence of disease, especially infectious disease. Therefore, if a traditional atua known to consume human beings existed prior to contact it would most likely have been in association with diseases such as cancer.

However, Ruatara's case deserves further mention as it has a pivotal role in the development of the beliefs. During a visit to England in 1809 Ruatara contracted tuberculosis. On his return from England he lived at Parramatta with Marsden for a while learning agricultural skills and around 1810 returned to New Zealand. By late 1814 Ruatara was suffering from a severe illness and was feverish with what appeared to be a 'violent cold' and other 'inflammatory symptoms'. (Nicholas 1817, p 151) Marsden recorded that Ruatara was very ill, with fever, foul tongue and violent bowel pain. (McNab 1908, p 390) Ten days later Ruatara was exhausted and in excruciating

According to Williams Dictionary, seventh edition, p 364, the phrase was used by Maori who believed

agony. His body was racked by paroxysms, possibly bowel spasms or coughing. Kendall wrote to Marsden, who had returned to Parramatta, on the 2nd March 1815 advising that Ruatara was worsening and he was complaining of shortness of breath and a weak and painful body. (McNab 1908, p 401) Ruatara died on March 3rd 1815 and Nicholas concludes by noting that Ruatara died of a bowel complaint and a stoppage in his breast, accompanied by a high fever and breathing difficulties. (Nicholas 1817, p 396)

In the final stages of Ruatara's illness, Maori told Kendall and Nicholas that in certain diseases of the lungs an atua entered them in lizard form and consumed their entrails. (Elder 1934, p 77) (Binney 1968, p 128n) (Elder 1934, p 86) When the entrails were all devoured he would be dead. (Nicholas 1817, p 170) The ancient Romans named the disease phthisis, meaning to waste away, and the English named the disease consumption. With tuberculosis it is not uncommon for the disease to spread to the bowels causing either bleeding or blockage of the bowel. Bleeding from the bowel and rapid wasting was probably the reason why Maori believed the atua was eating the insides of victims.

The conclusions that can be drawn from these points is that the entrail eating atua is probably not a traditional belief, but arose out of Maori desire to comprehend tuberculosis; out of ideas, vaguely understood, gleaned from missionaries, such as Marsden; and out of confusion generated by Kendall's attempts to express his beliefs and comprehension of Maori religion.

The belief that tuberculosis was caused by atua in lizard form continued into the 1820s with Cruise commenting that at the point that the person was believed to be incurable the person was believed to be afflicted by an atua. The Atua took possession of the person in the shape of a lizard and devoured the intestines. At this point no human assistance could be rendered and the person was separated from the village and left to die. (Cruise 1957, p 184) Cases of tuberculosis steadily increased through the 1820s and 1830s with periodic mention of European atua as the cause coming primarily from missionary records such as Butler in March 1822, (Barton

it was English and by Europeans who believed it was Maori.

1927, p 218 - 219) N Turner in September 1824, (McNab 1908, p 630) and Hobbs. (Williment 1985, p 36) Turangi, brother of Korokoro at Haumi was very unwell with tuberculosis and unable to move. He claimed his sickness came from Europeans and no sickness like this existed before their arrival. (Rogers 1961, p 76)

The belief in the entrail eating atua continued well into the late 1830s with Polack commenting that the lizard was a very virulent deity, (Polack 1838, p 229 - 230) and when Maori became ill it was because the atua was eating their entrails. (Polack 1838, p 332) In late 1834 Markham recorded that:

One reason for depopulation is the number of Pulmonary Complaints. They go off so suddenly, apparently in good Health. The old people believe that (Atua) God of the Parkiars Strangers is killing or eating the Mouries or Natives, and they see their strength decrease daily; some fancy a lizard in shape (Duetera) has got in their inside and is eating their entrails out. (Markham 1963, p 66)

The Old Men have an idea that the Atua of the parkeiahs is kiki-ing or eating up their people or nation, as they are aware that they are a diminishing people. (Markham 1963, p 66fn)

These statements by Markham are especially significant. As Markham did not have a good relationship with the missionaries, his statements independently record that Maori believed the entrail eating atua was of Parkeiah (European) origin that kiki (eat) the insides of people. More significantly they record that the 'lizard' that is responsible is Duetera. Ruatara, was also variously known as Tuatara or Duaterra. (Carrick 1903, p 164; Barton 1927, p 31) It would appear that the association is directly linked back to Ruatara, nearly twenty years after he had died and reinforces the thesis that entrail eating atua were a post contact belief associated with tuberculosis.

The 'Dying Race' Theory

By 1833 Reverend Davis believed that the native population was at risk of annihilation. (Coleman 1865, p 164) Markham also claimed in 1833 that pulmonary

disease was one of the causes of depopulation. (Markham 1963, p 66) (Markham 1963, p 46) Dr Tawell claimed Maori suffered a glandular affectation that was previously unknown to them. Tawell claimed hundreds were affected with the disease, both heathens and Christians. (Tawell 1908, p 119) Tawell's comment would suggest that Europeans also believed that tuberculosis had religious connotations, even though he was a doctor. This would reinforce the argument that the entailing atua arose out of a confusion of ideas regarding disease causation.

Seeking European Cures for European Diseases

With the ever increasing epidemics some Maori were influenced by missionary teachings and as the belief developed that God was responsible for the sicknesses Maori began to arrive at the missions in ever increasing numbers for European remedies. Butler recorded that Maori were coming to the missions requesting remedies from the missionaries as it was God that was making them sick. (Barton 1927, p 114) This belief pattern appears to have been emerging around 1821. Many of the missionaries made similar comments of Maori arriving at the missions for European remedies and foods, claiming that the diseases were caused by the European God. (Coates 1908, p 308) (Buller 1878, p 50) It would seem logical, therefore, for Maori to seek European cures. For example, in 1838 Wetekia brought a young man to Buller. He was trembling from head to foot with fear as he had eaten a sacred potato by mistake. He believed that this breach of tapu would cause an atua to enter him in the form of a lizard to eat his vital organs. He expected to die in three to four days. (Buller 1878, p 55) The fact that he turned to the missionaries for help would indicate that he believed his affliction was due to European atua or the European God. However, not all Maori believed the Europeans could offer any cures.

He Iwi Makutu

By the mid 1830s as the death rate from tuberculosis and other epidemics began to escalate, anger towards missionaries began to also escalate and their powers were being challenged. For instance, Rapu, who was suffering from tuberculosis, challenged Yate by saying 'If God can cure my body why does he not do so? And then I would believe what you tell me about my soul ... Let your God take away the pain out of my hand, and head, and side; let him make me well; and that will be such a sign, that everybody will then believe. What you say is too good for us and we

native men had better live as we are: your prayers require too much – more than we can do, if we tried'. Rapu died shortly after. (Yate 1835, p 294 - 296)

The developing mistrust encapsulated a perception that missionaries, there is no evidence this belief was attributed to other Europeans, were 'he iwi makutu', people capable of causing illness by magical or spiritual means. He iwi makutu were believed to be capable of inflicting illness on anyone they chose. While these beliefs began in mid 1815 through the 1820s they were gathering momentum. The missionaries were accused of causing George's illness in 1825 with their praying.

(Williment 1985, p 48) George died shortly after of tuberculosis. The beliefs were widespread by the early 1830s and escalated with the typhoid epidemic in 1832.

After the typhoid epidemic of 1832 Davis had written to Coates commenting that Maori believed the wrath of God caused the latest visitation of sickness on them because of their atrocious sins and he intended to sweep them away as a nation.

(Coates 1908, p 200) This epidemic in combination with the escalating rates of tuberculosis appears to have acted as a catalyst. At this time accusations began to be directed at the missionaries in force. Maori who did not believe in the powers of the European god were saying the Europeans were impostors and sorcerers and had the power of bringing sickness and destroying and killing them. (Coleman 1865, p 154)

(Coleman 1865, p 113) In June at Utuihu, Pomare's district, Maori claimed that the missionaries had put a makutu (curse) on them as they were all dying (Rogers 1961, p 248) and by November Warepoaka and Waikato were broadcasting that missionaries were he iwi matakū. (Rogers 1961, p 261) Henry Williams commented that they were saying this frequently. (Rogers 1961, p 248) Through 1833 a number of principal chiefs died at Thames (Rogers 1961, p 321) and others continued to die of various illnesses, but most especially of tuberculosis, at the Bay of Islands. Maori suspicion continued to mount and in April 1833 Te Morenga, who had been recently baptised became ill. Maori believed his illness was a result of him becoming a

Christian and warned Coleman that if he died they would avenge his death. (Coleman 1865, p 157) Te Morenga recovered but by September 1834 he was looking very ill and old. (Rogers 1961, p 391) While no record of his death has been found, he was suffering from tuberculosis and would have eventually died from it. Another to die during this period was Tohitapu, principal chief and tohunga at Haumi, who died of tuberculosis in July 1833. (Rogers 1961, p 323) He had had a competitive relationship with Henry Williams and had attempted to makutu Williams to no avail.

While Tohitapu had clung to some traditional beliefs he had not hesitated to come to the mission for European goods, either for himself, his tribe or for Hongi Hika during his period of convalescence. This may suggest that he also believed the diseases were European and was seeking European remedies. However, it is equally likely that this suggests he did not associated his own illness with European causation, as when he became ill he at first blamed a tohunga from Thames believing he was the victim of makutu and asked to be moved to the bush. (Rogers 1961, p 191) Later he was being cared for at the mission by Rawiri and Matiu for three days. He claimed he was getting worse and blamed the missionary karakia for preventing his recovery. (Rogers 1961, p 320) this suggests that Tohitapu remained enclosed in traditional ideas of causation, which he had extended to cover the European world. Tohitapu died in July 1833. (Rogers 1961, p 232)

Te Nakahi

By July 1833 a new doctrine had sprung up in opposition to the missionary teachings. The cult was named Nakahi, the serpent. (Rogers 1961, p 354) As discussed above the concept of the serpent was introduced into Maori society by Kendall as early as 1815, along with other biblical concepts. The Book of Genesis had been translated into Maori by 1833 and the serpent and the lizard appear to have been linked in Maori thought. The serpent is translated as Nakahi in Genesis 31. (Rogers 1961, p 354) As discussed above, the class of ground crawling creatures is ngarara, which includes lizards, centipedes and other creatures that crawl along the ground. While there are no serpents or snakes in New Zealand, the serpent fits into this ground crawling category. It is therefore possible that Maori would conflate the serpent and the ngarara resulting in a belief that tuberculosis was caused by a lizard consuming a person from within. This appears to have been a strong contributing factor in the development of the cult.

The principal leader of Nakahi was Papahurihia. (Binney 1968, p 322) Paphurihia believed that the bible was true but the missionaries had the wrong interpretation and had corrupted the word of God. (Wilson 1985) One of Papahurihia's strongest supporters, Waikato, was one of the missionaries strongest opponents. Waikato, amongst others, had been broadcasting that the missionaries were sorcerers for bringing epidemic disease amongst Maori. This opposition may well have had a part

to play in the development of the cult. Missionaries had attempted to use the epidemics to convert Maori to Christianity, with some eventual success. Wilson comments that many of the Maori who were attracted to the mission were suffering from disease.²⁷ However, Papahurihia, Waikato and others appear to have been reluctant to allow the missionaries that degree of power. Whatever Papahurihia's personal aspirations to leadership, the contextual evidence suggests that the incidence of infectious diseases, the increasing epidemics and the escalating cases of tuberculosis, coupled with missionaries claiming their God was responsible and Maori accusations that the missionaries were sorcerers, were all contributing factors in its development and strong following in the Bay of Islands and Hokianga.

In summary, there were a number of ideas that developed in Maori thought regarding the causation of diseases. The evidence suggests that in the first instance, Maori sought to explain disease by expanding traditional concepts. However, that was slowly superseded by the missionaries preaching that their God was the cause of disease and was punishment for their bad behaviour. There appears to have been a confusion of ideas for a period of time, but that eventually began to clarify. The concept that tuberculosis was caused by a specific atua was associated with the symptoms of the disease, traditional ideas that lizards were atua and Christian ideas that God punished sinners and the serpent was evil, or the devil if personified. As these ideas gathered momentum and Maori began to clarify their views on disease causation they began to blame the missionaries and turn against them. This in turn contributed to the development of the Nakahi cult.

²⁷ Yate included a number of letters from Maori at the mission in the back of his book. All of them described their illnesses and all of them are suffering various forms of tuberculosis.

CONCLUSIONS

The purpose of this thesis was to take a fresh look at the earliest available literature on Maori health to determine what impact introduced infectious diseases would have had on Maori societies in the period 1790 - 1840. My premise had been that with the lack of medical knowledge in the early 19th century, accounts of disease would have been inaccurate and would not have reflected the true incidence of endemic or introduced diseases in New Zealand. The early 19th century writers did not understand disease epidemiology particularly well nor did they know of micro-organisms. Therefore, 19th century accounts of disease would not offer an accurate reflection of the state of Maori health or the impact of introduced diseases. This was reflected in the findings that the impact of introduced diseases was greater than expected because the incidence of endemic disease was lower than anticipated. As a result of the low incidence of infectious disease in pre-contact Maori society there does not appear to have been a well developed belief system regarding infectious diseases, although Maori had a well developed belief system regarding misfortune in general.

Maori are of Polynesian origin and could have brought infectious diseases with them from Polynesia when they migrated to New Zealand. Therefore, chapter one of this thesis considered the migration of people through the Pacific and an analysis of diseases endemic in the Pacific offered an insight into any diseases that could have arrived from New Zealand. My research found that both the genetic and archaeological evidence agreed that the people of the Pacific are of South-East Asian origin. They moved out into coastal Melanesia and Polynesians originate from there. The founding groups into Polynesia were small and therefore the gene pool and the range of infectious diseases they could carry would also have been small. Most infectious diseases were or are zoonoses and there is a lack of suitable animal reservoirs for these diseases to have originated in most of Polynesia.

For that reason, the range of infectious diseases endemic to Polynesia are restricted to diseases brought by the initial immigrants and their animals. The chapter concluded that it would appear that the range of infectious diseases endemic to the Pacific is relatively small. There are a number of infectious diseases that are restricted to Melanesia and parts of Western Polynesia. These are all arthropod diseases and include arboviruses, malaria, scrub typhus and filariasis. These diseases did not

spread into Eastern Polynesia. Acute infectious diseases would not have survived the journey through the Pacific due to their short incubation period, short infectious period and their requirement for a large population to sustain them. There are a number of diseases that were clearly introduced into the Pacific by European explorers. These diseases included hepatitis A, enterobacteriaceae, ringworm, leprosy, tuberculosis, venereal diseases and lesser associated diseases such as eye diseases and tropical ulcers. The infectious diseases that may have been endemic to the wider Pacific, including Eastern Polynesia and New Zealand are limited to hepatitis B, herpes, and some strains of staphylococci and streptococci. However, these diseases may have been chronic, latent or sub acute diseases and their symptoms may not have been evident. Their incidence may be higher than we can assess.

Chapter two looked at the incidence of genetic and endemic diseases in New Zealand in the absence of introduced infectious diseases. This chapter established the benchmark against which the impact of introduced infectious diseases could be measured. A number of genetic diseases have been identified in New Zealand by archaeological evidence. These include gout, kidney disease, arteriosclerosis and also a possible case of adrenal cancer and a secondary cancer or histiocytosis - X. There is a lack of evidence for birth defects and deformities, which may be due to infanticide or loss of evidence as the bones of infants are soft and decay over time. However, there are no examples in the older skeletal samples. A few instances of deformities appear in the eyewitness evidence, but it is not always clear whether they are congenital or due to injury. The exception is incidence of clubfoot and harelip recorded in Queen Charlotte Sound. Of the infectious diseases that were possibly imported into New Zealand with the initial immigrants there is very little evidence for their existence, although they may have caused sub-clinical disease. There is tentative evidence for hepatitis B infection from Queen Charlotte Sound in European sailors who associated with Maori women. Alternatively, their illness could be toxin-mediated disease from shellfish. There is a lack of evidence for pneumonia, which neither proves nor disproves its existence. However, the observations of early travellers is congruent with a low incidence of disease, which meant a generally healthy population with reasonable physique and stature, initially good teeth that were affected by a harsh diet over time, healthy skin with good healing abilities. There was a lack of evidence for the existence of strains of staphylococci and streptococci. The

exception to this is evidence of abscesses in the jaws in skeletal samples, which may suggest the existence of *streptococci mutans*; however, more specific evidence is needed. There is a lack of evidence for infection after tattooing, although a number of deaths could have arisen as a result of severe inflammation caused by an immune system reaction. The population included a number of old people who were aged between 60 - 70, however, the exact number of old people is not known. The conclusions that could be drawn from this chapter was that the incidence of endemic infectious disease was very low.

In chapter three the impact of introduced acute infectious diseases was considered. Having lived in isolation and with a low incidence of infectious diseases, the impact of introduced infectious diseases would have had a significant impact on morbidity and mortality, although the exact impact is difficult to measure due to the sporadic nature of the observational evidence. However, during certain epidemics there appears to have been a significant death rate. Certain diseases would have had greater impact than other diseases. For instance, diseases, such as childhood diseases, that inferred immunity on the survivors after first contact might not have had the same impact on the same people the second time around. There does not appear to be hugely significant numbers of deaths from these epidemics, although some deaths did occur. The exception to this was the epidemic of measles in the South Island in 1835 followed by an influenza epidemic in 1837 - 8. According to Maori and whalers alike, the impact on the Maori population was devastating and led to the establishment of whaling stations in coastal areas, which had been initially opposed by local Maori, who were no longer able to offer any resistance. The evidence suggested that influenza epidemics had varying impacts; this would suggest varying degrees of virulence. According to Maori in Taranaki the epidemic of catarrh in 1820 killed thousands. Whether this is a true reflection of the death rate is difficult to measure; however, the epidemic did bring about a number of deaths at Hauraki and led to the abandonment of the kainga. While the estimate of thousands may be a little high, it does suggest that Maori were quite overwhelmed by the enormity of the disease. This is understandable for a people who had not experienced epidemics before. An epidemic of typhoid and an epidemic of influenza in the 1830s do suggest a quite high death rate. However, this may also be due to the increasing incidence of illness and the reduced opportunity for the population to recover. The early

epidemics, probably of typhus before 1810, indicate that Maori believed that these diseases were European in origin and caused by European atua. This claim appears with increasing frequency throughout the observational literature and suggests that Maori had not experienced these diseases before and therefore did not have a well developed belief system regarding acute infectious disease. This is to be expected in a society that had not experienced these epidemics before.

Chapter four explored the incidence and impact of introduced chronic diseases, namely tuberculosis and venereal diseases. These diseases had the greatest impact, especially at the Bay of Islands, because of their longevity. Venereal disease was prevalent by the 1830s, especially syphilis. The disease had a significant impact at first, and struck with a virulence that would indicate a fairly new disease. This would suggest that the disease first introduced by the early French and English explorers in the 1770s did not become established and was reintroduced by later whalers and traders. The evidence from Murihiku, slim though it is, also suggests the disease was virulent there as well, although the incidence is difficult to assess. As with the acute infectious diseases discussed above, Maori believed that venereal diseases were caused by European atua. The disease appears to have affected Maori women more than men because of the sex industry, however, a number of methods that reduced the incidence of disease, regardless of whether these methods were used deliberately or not. The men put the sex industry women under tapu and would not have any relationships with them while they were working the ships. The women used a vapour bath of steamed herbs to alleviate symptoms; both of these practices would have reduced the incidence of the disease. The practice of infanticide or abortion, which was widely practised by the ships' women, would have reduced the incidence of congenital syphilis considerably. Tuberculosis was the most significant disease. It first appears in the literature from around 1808 and the first recorded cases were amongst those who had travelled outside of New Zealand to Australia or England. The disease appears to have manifested itself in many forms, although the pulmonary and scrofulous forms appear to have been the most frequent. Other diseases, especially of a respiratory nature, such as influenza exacerbated the incidence of tuberculosis. It is therefore difficult to determine the ultimate cause of death in these cases, but ultimately the tuberculosis sufferer would succumb to this disease even if he or she had survived an attack of influenza. By the early 1830s deaths from

tuberculosis were beginning to escalate and by the 1830s Europeans, especially the missionaries, were beginning to claim Maori were a dying race.

Chapter five explored the impact the diseases had had on Maori beliefs. In the preceding chapters conclusions had been drawn that there was a very low incidence of infectious diseases, prior to the introduction of infectious diseases from the outside world. As a result of the very low incidence of infectious diseases Maori would have had no need for a well developed belief system regarding infectious diseases, however, with the arrival of acute infectious diseases, such as whooping cough and influenza, and chronic infectious diseases, such as venereal disease, an explanation was required as to the cause of these diseases. As Maori already had a well developed system of beliefs to explain misfortunes, their system appears to have been expanded to include European diseases. It would seem logical that if Maori recognised that the diseases were arriving with the Europeans, then Maori would believe the diseases must be caused by European atua. Traditionally atua had been familial, focussing their attentions and punishments on family members or others who could cause them harm. The most malevolent form of atua were infant spirits known as kahukahu. In the Bay of Islands the introduction of infectious diseases necessitated some change, as existing beliefs did not cater to the new diseases. A number of theories of disease causation sprung up in the region. Some believed that their atua were angry and some believed that it was the European gods. A theory of disease transmission developed that claimed the diseases were arriving with the vessels or with Europeans. This belief that European atua were the cause of disease was applied most especially to tuberculosis and appears to have started with Ruatara. The idea developed that the disease, or the atua as the case may be, came in the form of a lizard, which consumed the person from the inside. Given that tuberculosis causes rapid emaciation and has been called consumption it is not surprising that Maori sought to explain this rapid wasting away of their friends and relatives. As the beliefs developed, a reactionary force was also developing with a group of Maori claiming that the missionaries were the iwi makutu and were destroying the people with their praying. This appears to have been an underlying philosophy in the development of the cult of Papahurihia, as the cult arose in the wake of a typhoid epidemic and as deaths from tuberculosis were escalating, and as Europeans, especially missionary were claiming they were a dying race.

In conclusion, while it is difficult to assess exact morbidity and mortality from the observational evidence, it is clear that the diseases that were introduced after contact had a significant impact on the population of New Zealand, most especially in the upper North Island where contact was more frequent. As there had been a low incidence of infectious disease prior to contact, one impact would have been reasonably significant, however, the diseases came in increasing numbers as the outside world intruded. Maori attempts to understand the disease were reflected in the growing debate that developed regarding causation, most especially the idea that a lizard was consuming their entrails. As previously discussed, this idea is usually mentioned in conjunction with cases of tuberculosis and suggests that it is reflective of the disease itself, rather than reflective of any traditional Maori beliefs about disease in general.

ACKNOWLEDGEMENTS

There are many people to thank in association with this thesis. Without the help, support, encouragement and feed-back that I have received the researching and writing of this thesis would have been all the more difficult. I therefore, thank every one who has crossed my path and aided my work over the past year, but I would like to thank a number of individuals personally.

The first of these is my son, Dylan Tosca. He is my medical misadventure turned medical miracle and the inspiration behind this thesis. He has taught me how to turn disabilities into challenges to be overcome. May his journey be paved with a thousand blessings.

I also thank his kuia, Wendy Wadworth, who has shown us the meaning of whanau and has supported us through all the hard times and now the good times. Aroha ki a koe, e hoa.

I thank also my koro, Arthur and his wife Jean, who also supported us and took me under their wings. Thank you for your love and guidance. While I know you watch over me your presence in this realm is sadly missed.

Lisa, my dearest friend, you understand what this has taken more than anybody else and your support has also been much appreciated.

To my supervisor, Lyndsay Head, who is still frantically checking my thesis as I write this, thank you for your guidance, encouragement and feed back. This thesis will be all the better for your help.

To all the other staff at the University, especially the staff in the Macmillan Brown Library, and to all those other people who have assisted, supported and encouraged me, I also give thanks.

There were many things that went wrong in the last four weeks of writing this thesis. All of my documents, including this thesis, were erased during a backup after the installation of a new server. The floppy disk backup I had at home was several days old and much time was lost in restoration. In the final few days chapters that had been checked by my supervisor were stolen from the department following a number of unauthorised entries into my office. All of this resulted in significant setback and this is reflected in the final chapter, which had to be handed in incomplete. I therefore apologise to any readers of this thesis and give special thanks to Anna King who helped me in my last minute rush. I also thank Owen in security for his sensitive handling of the theft.

BIBLIOGRAPHY

- Anderson, A. 1989. The Last Archipelago: 1000 Years of Maori Settlement in New Zealand. Towards 2000. A. A. et. al. Wellington.
- Anderson, A. 1998. The Welcome of Strangers: An Ethnohistory of Southern Maori 1650 - 1850. Dunedin, University of Otago Press.
- Barton, R. J., Ed. 1927. Earliest New Zealand: The Journals and Correspondence of the Reverend John Butler 1819 - 1823. Masterton, Palamontain & Petherick, General Printers.
- Batra, V. 2002. "Tuberculosis." eMedicine Journal 2002 April.
- Beaglehole, J. C. 1955. The Journals of Captain James Cook: The Voyage of the Endeavour 1768 - 1771. Vol 1, Cambridge, Cambridge University Press.
- Beaglehole, J. C. 1961. The Journals of Captain Cook, The Voyage of the Endeavour 1768 - 1771. Vol 2, Cambridge, Cambridge University Press.
- Beaglehole, J. C. 1962. The Endeavour Journal of Joseph Banks, 1768 - 1771. Vol 1, Sydney, Angus and Robertson Limited.
- Beaglehole, J. C. 1962. The Endeavour Journal of Joseph Banks, 1768 - 1771. Vol 2, Sydney, Angus and Robertson Limited.
- Begg, A. C. B., N C 1973. Port Preservation. Christchurch, Whitcoulls Limited.
- Bellwood, P. S. 1989. The Colonization of the Pacific: Some Current Hypotheses. The Colonization of the Pacific: A Genetic Trail. A. V. S. S. Hill, S W. Oxford, Clarendon Press: 1 - 59.
- Bennett, G. 1860. Gatherings of a Naturalist in Australasia: Being Observations Principally on the Animal and Vegetable Productions of New South Wales, New Zealand and some of the Austral Islands. London, John Voorst.

- Bennett, G. 1883. "Notes on Diseases Prevailing among the New Zealanders and Other Inhabitants of the Polynesian Islands, with Some Account of their Mode of Treatment and their Superstitions connected Therewith." Australasian Medical Gazette 31: 1 - 5.
- Best, E. 1924. Maori Religion and Mythology. Wellington, W A G Skinner, Government Printer.
- Binney, J. 1968. The Legacy of Guilt: A Life of Thomas Kendall. Auckland, Oxford University Press.
- Bright, D. J. 1841. Handbook for Immigrants, and others, Being a History of New Zealand, Its State and Prospects, Previous and Subsequent to the Proclamation of Her Majesty's Authority. London, Henry Hooper.
- Buller, J. 1878. Forty Years in New Zealand. London, Hodder & Stoughton.
- Calder, A. 2001. Old New Zealand and Other Writings, F E Maning. London, Leicester University Press.
- Carleton, H. 1948. The Life of Henry Williams: Archdeacon of Waimate. Wellington, A H & A W Reed.
- Carrick, R. O. 1903. Historical Records of New Zealand South Prior to 1840. Dunedin, Otago Daily Times and Witness Newspapers Co. Limited.
- Chandrosoma P & Taylor C. R. 1995. Concise Pathology. New Jersey, Appleton & Lange.
- Coates, D. B., J., 1908. Brought from the Lords 7th August 1838: Report From the Select Committee of the House of Lords, Appointed to Inquire into the Present State of the Islands of New Zealand, and the Expediency of Regulating the Settlement of British Subjects Therein; with the Minutes of Evidence Taken Before the Committee and an Index Thereto, Ordered by the House of Commons to be Printed 8 August 1838, British Parliamentary Papers: Colonies New Zealand.

- Coleman, J. N. 1865. A Memoir of the Reverend Richard Davis: For Thirty-nine Years a Missionary in New Zealand. London, James Nisbet & Co.
- Craik, J. L. 1830. The New Zealanders. London, Charles Knight.
- Cruise, R. A. 1957. Journal of a Ten Months Residence in New Zealand, 1820. Christchurch, Pegasus Press.
- Cumpston, J. H. L. 1989. Health and Disease in Australia : A History. Canberra, Australian Government Publishing Service.
- Diamond, J. 1998. Guns, Germs and Steel: A Short History of Everybody for the Last 13,000 Years. United Kingdom, Vintage Random House UK Limited.
- Dieffenbach, E. 1843. Travels in New Zealand: With Contributions to the Geography, Geology, Botany and Natural History of that Country. Vol 1, London, John Murray, Ablemarle Street.
- Dieffenbach, E. 1843. Travels in New Zealand: With Contributions to the Geography, Geology, Botany and Natural History of that Country. Vol 2, London, John Murray, Ablemarle Street.
- Dillon, P. 1829. Narrative and Successful Results of a Voyage in the South Seas Performed by Order of the Government of British India to Ascertain the Actual fate of La Perouse Expedition. London, Hurst, Chance & Co.
- Dumas, A. M., F, Ed. 1937. The Whalers. London, Hutchinson & Co Publishers Ltd.
- Earle, A. 1909. A Narrative of Nine Months Residence in New Zealand in 1827. Christchurch, Whitcombe & Tombs Limited.
- Elder, J. R. 1934. Marsden's Lieutenants. Dunedin, Otago University Council.

- Flatt, J. 1908. Brought from the Lords 7th August 1838: Report From the Select Committee of the House of Lords, Appointed to Inquire into the Present State of the Islands of New Zealand, and the Expediency of Regulating the Settlement of British Subjects Therin; with the Minutes of Evidence Taken Before the Committee and an Index Thereto, Ordered by the House of Coomons to be Printed 8 August 1838, British Parliamentary Papers: Colonies New Zealand.
- Forster, G. 1777. A Voyage Around the World in his Britannic Majesty's Sloop Resolution, Commanded by Captain James Cook, During the Years 1772, 1773, 1774 and 1775. London.
- Gluckman, L. K. 1976. Tangiwai: A Medical History of New Zealand Prior to 1860. Christchurch, Whitcoulls Limited.
- Hanham, D. 2001. Maori Health and Introduced Disease: 1769 - 1790. Unpublished Research Paper. Christchurch.
- Hansen, E. A. C., B A 2002. "Typhus." eMedicine Journal 33: 10.
- Harrop, E., M.D. 2002. "Leprosy." eMedicine Journal 34: 1 - 14.
- Hemplemen, G. 1911. The Piraki Log E Pirangi Ahau Koe or Dairy of Captain Hempleman with Introduction, Glossary, Illustrations and Map. London, Henry Froude, Oxford University Press.
- Herchline, T., M. D. 2002. "Staphylococcal Infections." eMedicine Journal 32: 1 - 13.
- Houghton, P. 1980. the First New Zealanders. Auckland, Hodder & Stoughton.
- Howe, K. R. 1984. Where the Waves Fall: A New South Sea Islands History from First Settlement to Colonial Rule. Sydney, George Allen & Unwin.
- Jawetz E. Melnick J. L. & Adelberg E. A. 1970. Review of Medical Microbiology. California, Lange Medical Publications.

Kuhn, G. 2001. "Pneumonia, Viral." eMedicine Journal.

Markham, E., 1963. New Zealand or Recollections of it. Wellington, R E Owen, Government Printer.

Marshall, W. B. D. 1834. A Personal Narrative of Two Visits to New Zealand in HMS Alligator 1834. London, James Nisbet & Co.

McKay, A. 1873. A Compendium of Official Documents Relative to Native Affairs in the South Island. Wellington, Government Printer.

McNab, R. 1908. Historical Records of New Zealand. Wellington, Whitcombe & Tombs Ltd.

McNab, R. 1909. Murihiku: A History of the South Island of New Zealand and the Islands Adjacent and Lying to the South from 1642 to 1835. Wellington, Whitcombe & Tombs Ltd.

McNab, R. 1914. From Tasman to Marsden: A History of Northern New Zealand From 1642 to 1818. Dunedin, J Wilkie & Co Ltd.

McNab, R. 1914. Historical Records of New Zealand. Wellington, Whitcombe & Tombs Ltd.

Meltzer, M. S., M.D. & Nacy, C A, PhD 2002. "Cutaneous Tuberculosis." eMedicine Journal 32: 1 - 15.

Miles, J. 1997. Infectious Diseases: Colonising the Pacific. Otago, University of Otago Press.

Montgomerie, J. Z. 1988. "Leprosy in New Zealand." Journal of the Polynesian Society 972: 116 - 146.

Montgomery, J. 1831. Journal of the Voyages and Travels by the Reverend Daniel Tyerman and George Bennet Deputed from the London Missionary Society to Visit their

Various Stations in the South Sea Islands, Between the Years 1821 - 1829. London, Frederick Westley & A H Davis.

Mudur, G. 2001. "Indian Scientists warn of "mutant measles" virus." British Medical Journal 32224 March: 693.

Nicholas, J. L. 1817. Narrative of a Voyage to New Zealand, Performed in the Years 1814 and 1815, In Company with the Reverend Samuel Marsden, Principal Chaplain of New south Wales. Vol 1, London, James Black and Son.

Nicholas, J. L. 1817. Narrative of a Voyage to New Zealand, Performed in the Years 1814 and 1815, In Company with the Reverend Samuel Marsden, Principal Chaplain of New south Wales. Vol 2, London, James Black and Son.

Nicholas, J. L. 1908. Brought from the Lords 7th August 1838: Report From the Select Committee of the House of Lords, Appointed to Inquire into the Present State of the Islands of New Zealand, and the Expediency of Regulating the Settlement of British Subjects Therein; with the Minutes of Evidence Taken Before the Committee and an Index Thereto, Ordered by the House of Commons to be Printed 8 August 1838, British Parliamentary Papers: Colonies New Zealand.

Ollivier, E. 1986. Early Eyewitness Accounts of Maori Life 3 and 4: Extracts from the Journals of the Ships Recherche, Esperance and Coquille, 1793 and 1824 - McMillan Brown. Wellington, Alexander Turnbull Library Endowment Trust.

Orbell, M. 1995. The Illustrated Encyclopedia of Maori Myth and Legend. Christchurch, Canterbury University Press.

Polack, J. S. 1838. New Zealand: Being a Narrative of Travels and Adventures During a Residence in that Country Between the Years 1831 and 1837. Vol 1, London, Richard Bentley.

- Polack, J. S. 1838. New Zealand: Being a Narrative of Travels and Adventures During a Residence in that Country Between the Years 1831 and 1837. Vol 2, London, Richard Bentley.
- Polack, J. S. 1908. Brought from the Lords 7th August 1838: Report From the Select Committee of the House of Lords, Appointed to Inquire into the Present State of the Islands of New Zealand, and the Expediency of Regulating the Settlement of British Subjects Therein; with the Minutes of Evidence Taken Before the Committee and an Index Thereto, Ordered by the House of Commons to be Printed 8 August 1838, British Parliamentary Papers: Colonies New Zealand.
- Polack, J. S. 1976. Manners and Customs of the New Zealanders with Notes Corroborative of Their Habits, Usages &c and Remarks to Intending Emigrants. Vol 1, Christchurch, Capper Press.
- Polack, J. S. 1976. Manners and Customs of the New Zealanders with Notes Corroborative of Their Habits, Usages &c and Remarks to Intending Emigrants. Vol 2, Christchurch, Capper Press.
- Porter, F., & MacDonald, C, 1996. My Hand Will Write What My Heart Dictates: The Unsettled Lives of Women in Nineteenth Century New Zealand as Revealed to Sisters, Family and Friends.
- Porter, R. 1997. The Greatest Benefit to Mankind: A Medical History of Humanity. New York, W Norton & Co, Inc.
- Richards, R. 1995. Murihiku Reviewed.
- Rogers, L. M. 1961. The Early Journals of Henry Williams: Senior Missionary in New Zealand of the Church Missionary Society 1826 - 1840. Christchurch, Pegasus Press.
- Ryburn, H. J. 1979. Te Hemara: James Hamlin 1803 - 1865: Friend of the Maoris. Dunedin, John McIndoe Ltd.

- Savage, J. 1939. Some Account of New Zealand; the Bay of Islands, and surrounding country; with a description of the Religion and Government, Language, Arts, Manufacturers, Manners, and Customs of the Natives. Savage's Account of New Zealand in 1805 Together With Schemes of 1771 and 1824 for Commerce and Colonization: Reprinted with Notes. A. D. McKinlay. Wellington, L T Watkins Limited.
- Schleiss, M. R., M. D. 2002. "Streptococcal Infection, Group A." eMedicine Journal 34: 1 - 21.
- Serjeantson, S. W. 1989. The Colonization of the Pacific: The Genetic Evidence. The Colonization of the Pacific: A Genetic Trail. A. V. S. S. Hill, S W. Oxford, Clarendon Press: 286 - 294.
- Sharma, S. 2002. "Pneumonia, Bacterial." eMedicine Journal.
- Shortland, E. L. 1856. Traditions and Superstitions of the New Zealanders. London, Longman, Brown, Green, Longmans & Roberts.
- Shortland, E. L. 1974. The Southern Districts of New Zealand, with Passing Notices of Customs of the Aborigines. Christchurch, Capper Press.
- Smith, S. P. 1910. History and Traditions of the Maoris of the West Coast North Island of New Zealand prior to 1840. New Plymouth, Polynesian Society.
- Starke, J. 1986. Journal of a Rambler: The Journal of John Boulton. Auckland, Alexander Turnbull Library Endowment Trust, Oxford University Library.
- Straubel, C. R. 1954. The Whaling Journal of Captain W B Rhodes: Barque Australian of Sydney 1836 - 1838, Whitcombe and Tombs Limited.
- Swartout, H. O. 1957. Modern Medical Counsellor: A Practical Guide to Health. Victoria, Signs Publishing Company.

- Tawell, J. D. 1908. Brought from the Lords 7th August 1838: Report From the Select Committee of the House of Lords, Appointed to Inquire into the Present State of the Islands of New Zealand, and the Expediency of Regulating the Settlement of British Subjects Therein; with the Minutes of Evidence Taken Before the Committee and an Index Thereto, Ordered by the House of Commons to be Printed 8 August 1838, British Parliamentary Papers: Colonies New Zealand.
- Taylor, R., Rev., 1974. Te Ika a Maui: New Zealand and its Inhabitants. Wellington, A H & A W Reed Ltd.
- Tremewan, C. 2002. Traditional Stories from Southern New Zealand. Christchurch, Macmillan Brown Centre for Pacific Studies, University of Canterbury.
- Turnball, J. 1813. A Voyage Round the World in the Years 1800, 1801, 1802, 1803 and 1804; in which the Author Visited Madeira, the Brazils, Cape of Good Hope, the English Settlements of Botany Bay and Norfolk Island and the Principal Islands in the Pacific Ocean with a Continuation of their History to the Present Period. London, A Maxwell, Bell Yard, Temple Bar.
- Waseem, M. 2002. "Syphilis." eMedicine Journal 34.
- Watkins, J. 1908. Brought from the Lords 7th August 1838: Report From the Select Committee of the House of Lords, Appointed to Inquire into the Present State of the Islands of New Zealand, and the Expediency of Regulating the Settlement of British Subjects Therin; with the Minutes of Evidence Taken Before the Committee and an Index Thereto, Ordered by the House of Commons to be Printed 8 August 1838, British Parliamentary Papers: Colonies New Zealand.
- Wilkinson, F. 1908. Brought from the Lords 7th August 1838: Report From the Select Committee of the House of Lords, Appointed to Inquire into the Present State of the Islands of New Zealand, and the Expediency of Regulating the Settlement of British Subjects Therein; with the Minutes of Evidence Taken Before the Committee and an Index Thereto, Ordered by the House of Commons to be Printed 8 August 1838, British Parliamentary Papers: Colonies New Zealand.

Williams, F. W. 1939. Through Ninety Years, 1826 - 1916, Life and Work Among the Maoris in New Zealand. Auckland, Whitcombe & Tombs.

Williams, W. 1867. Christianity Among the New Zealanders. London, Seeley, Jackson & Halliday.

Williment, T. M. I. 1985. John Hobbs, 1800 - 1883: Wesleyan Missionary to the Nga Puhi Tribe of Northern New Zealand. Wellington, V R Ward, Government Printer.

Wilson, O. 1985. From Hongi Hika to Hone Heke. Dunedin, John McIndoe Ltd.

Wright, O. 1950. New Zealand 1826 - 1827 from the French of Dumont D'Urville: An English Translation of the Voyage de L'Astrolabe in New Zealand Waters. Wellington, Wingfield Press.

Yate, W. R. 1835. An Account of New Zealand and the Formation and Progress of the Church Missionary Society's Mission in the Northern Island. London, Seeley and Burnside.